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PRIMARY SPLENOMEGALY—ENDOTHELIAL HYPERPLASIA OF THE SPLEEN—TWO CASES IN CHILDREN—AUTOPSY AND MORPHOLOGICAL EXAMINATION IN ONE.[1]

By David Bovaird, Jr., M.D.,
of New York,
Pathologist to the Foundling Hospital; Clinical Instructor in Diseases of Children, University and Bellevue Hospital Medical College.

The cases herein reported represent, in the writer's belief, an affection hitherto unrecognized. But two complete reports of similar cases are to be found in literature, and in both of these it can be clearly shown that the pathological findings were wrongly interpreted. The cases were of such interest that they were kept under observation for a period of nearly three years, during which time no definite conclusion as to the nature of the affection present could be reached.

It was only the light afforded by the autopsy and subsequent morphological examination in one of the cases that made clear the nature of the morbid process.

The histories are as follows:

The children were first seen in November, 1896, and were referred to the Presbyterian Hospital, where the following observations were made:

Case I.—M. K., aged three years. (See Fig. 1.) Clinical record only.

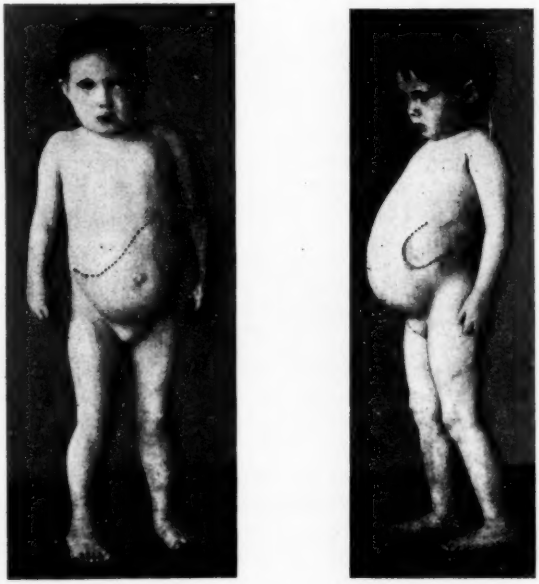
Family History. Father and mother both living and well. Mother has borne ten children, of whom one was still-born at eight months, four died during infancy from "summer complaint," and five are living.

[1] Awarded the Alumni Association Prize of the College of Physicians and Surgeons (Columbia University), 1900.

Of the living, three boys, aged respectively eleven years, eight years, and five years, are healthy and vigorous, presenting no traces of disease. The patient and her sister, described below as Case II., are the only invalid members of the family. It is to be noted that the boys stand in age between the sisters. The deaths in infancy above recorded were not successive, but occurred between the births of children that survived. The mother is free from any traces of disease. The father was not seen, but was reported perfectly well.

Previous History. The only ailment has been occasional attacks of toothache, with some swelling of the right side of neck.

FIG. 1.



CASE I. M. K., aged three years.
Dotted lines indicate margins of liver and spleen.

Present Illness. For about one year the mother has noticed that this child's abdomen has been growing prominent, the enlargement steadily increasing. She has also observed that there was a hard mass on the right side of the abdomen, as well as upon the left. Otherwise the child has been quite well. No chills or fever; no headache; no sore throat; no swelling of the feet; no disease of skin or bones. For three weeks she has had a slight cough, but has not been sick with it. The appetite is good, the bowels regular. On admission to hospital: temperature, 99.8°; pulse, 108; respiration, 36. Urine, sp. gr., 1031; acid, trace of albumin; leucocytes, but no casts.

Physical Examination. Child is of normal stature, well nourished, and not anæmic; the skin is rather dusky; tongue moist and clean, the teeth normal. Lungs: Over the whole right lung abundant sibilant and sonorous, fine and coarse mucous râles. Over the left, a few râles of similar character; otherwise normal. Heart: Apex in fourth space, three inches from median line. Sounds and action normal. Liver dulness extends from fourth space to three inches below free border of ribs in midclavicular line, where edge can be distinctly felt; edge is moderately hard and sharp. Spleen: Emerges from beneath ribs in midclavicular line, extends downward and inward to within half an inch of median line at umbilicus, the lower end being on a level with the anterior superior spine of the ilium. The edge is sharp and hard; the notch in anterior margin is felt one inch below the costal margin. Abdomen is otherwise normal. Extremities normal. Superficial lymph nodes: In each axilla one node, size of a bean, can be felt. Both sets of inguinal nodes are slightly enlarged. The nodes are not painful or tender, and are freely movable. Tonsils, both much enlarged.

Examination of Blood. Red cells, 4,400,000; white cells, 9000; hæmoglobin, 75 per cent. No malarial organisms. During her stay in hospital, from November 28, 1896, to December 22, 1896, the child seemed quite well. The chest signs cleared up and the cough ceased. She gained one and three-quarters pounds in weight; weighed thirty-two and three-quarters pounds at discharge. Throughout her stay Fowler's solution was given in increasing doses until she was getting fourteen minims per day.

The physical examination at discharge, with the exception of the signs of bronchitis, was as above given. This child was also seen on March 30, 1897. At that time the liver was as already described. The spleen had apparently increased somewhat in size, as it then reached the median line and extended some distance (about an inch and a half) below the umbilicus. Otherwise the child was perfectly well and happy. An examination of the blood gave: Red blood-cells, 4,800,000; hæmoglobin, 65 per cent. The white cells were not counted, but studied in stained smears they showed no relative increase and no abnormalities in form. The red cells were also normal in form.

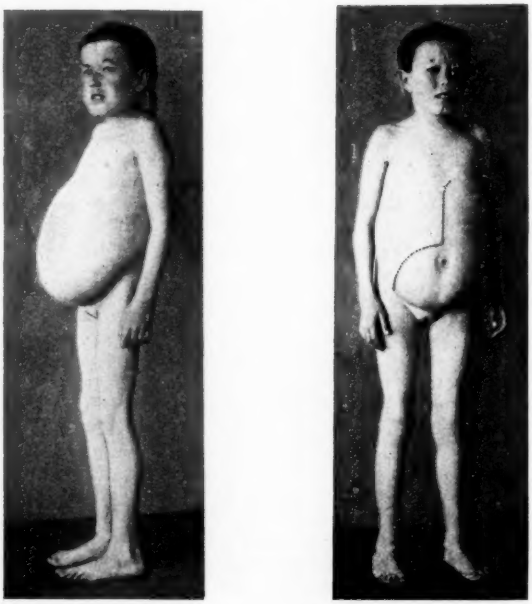
July 26, 1899. For some time, the mother says, the child had been failing. Her appetite has remained good. She eats a great deal of meat, and eggs, and sugar, drinks much coffee, but will hardly touch vegetables. The bowels are constipated. Her nose bleeds frequently and very freely. She always has a cough; never vomits; has lost flesh very decidedly. During the past summer her face and hands have become bronzed.

Examination at this time, aged six years, four years after onset. Across the bridge of the nose and on cheeks and hands there is a diffuse bronzing of the skin, similar to that observed in her sister, Case II. The color of the mucous membranes is good; the tongue is normal; the teeth are decayed; the gums normal; the lungs and heart are normal. The abdomen looks much more prominent than when first seen; measures twenty-three and one quarter inches at umbilicus. The superficial veins of the abdomen are engorged. The liver extends from the sixth rib in the axillary line to within an inch and a half of the umbilical line. The left lobe is apparently the more enlarged, filling the whole epigastrium. There has evidently been some prolapse of the organ since the

last examination. The spleen now extends into and fills the right iliac fossa. Between the enlarged liver and spleen there is a narrow space about two inches in width, which is not occupied by the solid viscera. The extremities have lost their full, round appearance, and are now thin. The inguinal, axillary, and cervical lymph nodes are all slightly enlarged, but feel soft rather than hard.

The examination of the blood at this time showed: Red cells, 4,180,000; white cells, 14,000; hæmoglobin, 62 per cent. A differential count of the white cells showed: Polynuclears 57 5 per cent., normal 62–70 per cent.; mononuclears, large, 29 per cent., normal 4–8 per cent.; small

FIG. 2.



CASE II. D. K., aged thirteen years.
Dotted line indicates anterior margin of spleen.

12.5 per cent., normal 35 per cent.; eosinophiles 1 per cent., normal ½–4 per cent.

A recent note from the mother (December, 1899) states that the child has not improved in any way, but is still able to attend school.

CASE II.—D. K., aged thirteen years. (See Fig 2.) Clinical record, with autopsy, etc.

Family History. As above.

Previous Personal History. At five years of age child had measles; at seven an eruption, limited to the scalp, and consisting of large areas covered by scabs, which lasted three or four months. No chills or

fever; no headache; no sore throat; no cough; no vomiting; no swelling of the feet. No other illness beyond that to be recorded.

Present Illness. When she was about three years old her mother noticed that the abdomen was getting larger than normal, and since that time there has been a progressive increase in its size. Soon after noting the protuberance of the abdomen the mother found that there was a hard mass in the left side, and has observed that this mass has increased in size with the swelling of the abdomen. The mother has also noted the brownish pigmentation of the skin, especially across the nose, and has thought that the extremities have become thinner of late. The child has complained of little discomfort from the enlarged abdomen, has been a little short of breath, but otherwise has been quite well. The appetite is good, the bowels regular.

On Admission to Hospital. Temperature, 99.8°; pulse, 96; respiration, 24.

Urine. Specific gravity, 1024, 2 per cent. albumin, moderate number of granular and epithelial casts and leucocytes.

Physical Examination. Child is rather small in stature for her years; is fairly nourished. The skin generally is rather dusky, and across the nose and underneath the eyes and upon the upper lip there is a decided brownish pigmentation. She lies from choice upon the left side. Tongue is moist and clean; teeth normal. Heart: Apex in fourth space, two and a half inches from median line; action regular and of good force; no murmurs. Lungs: Apparently normal; no signs of compression. Liver: Dulness extends from fifth space to free margin of the ribs; edge cannot be felt. Abdomen: Protuberant, girth at umbilicus thirty-one and a quarter inches; superficial veins somewhat dilated. Three-quarters of an inch to the left of the median line there emerges from beneath the ribs a solid mass, with a firm, rounded edge which extends downward in the median line to a point about one inch above the umbilicus, and thence passes obliquely across to the right anterior superior spine of the ilium. The mass, therefore, fills the whole of the left side of the abdomen and most of the lower half of the right side. Just above the umbilicus there is a deep notch in the border of the mass, in which a finger can easily be laid. The mass feels hard and smooth; is not tender; in every way presents the characters of an enlarged spleen. Extremities are normal. Superficial lymph nodes; slight enlargement of right inguinal; moderate enlargement of both submaxillaries; one palpable node in each axilla; no enlargement of the epitrochlear nodes. Tonsils: Both moderately enlarged.

An examination of the blood showed: Red blood-cells, 2,880,000; white cells 4000; hæmoglobin, 60 per cent. No malarial organisms.

During her stay at the hospital, from November 28, 1896, to December 22, 1896, the child was up and about, playing naturally with the other children. There was occasionally slight constipation, easily relieved. Later examination of the urine showed a specific gravity of 1030, an acid reaction, only a trace of albumin, and no casts. Voided about twenty-four ounces per day. While in the hospital she was given Fowler's solution in gradually increased doses, until at time of discharge she was taking eighteen minims per day.

The physical examination made just previous to discharge corresponded exactly to that above recorded. There was no change in the viscera. Temperature was normal throughout her stay in hospital.

Weighed seventy pounds at discharge, a gain of four and three-quarter pounds.

After spending four months at home, the patient was seen on March 30, 1897, and again examined. The splenic enlargement was practically unchanged. The mother reported that since leaving the hospital the general health has been good; that, although somewhat short of breath, the child had attended school, and had been cheerful and happy.

The examination of the blood at this time showed: Red cells, 2,800,000; hæmoglobin, 45 per cent. The white cells were not counted, but studied in stained smears. They showed no relative increase in number and no abnormalities of form. The red cells were also normal in form.

In the interval since leaving the hospital specific treatment, both in the form of inunctions of blue ointment and the internal administration of the bichloride of mercury and potassium iodide had been tried without effect.

The condition of this child remained practically the same. She did not grow in stature or gain in weight. The mental condition after three years (at the age of sixteen years) was that of a child. The possibilities of medical treatment having been exhausted without any relief, the mother asked to have an operation undertaken, as it was evident that the child was slowly but surely going down under the influence of her disease. She was admitted to the hospital May 15, 1899.

Examination at the age of sixteen years, thirteen years after onset. Child fairly well-nourished. Face and hands deeply bronzed, in a manner somewhat similar to that seen in Addison's disease. Tongue, clean. Lungs, normal, Heart, systolic murmur over the pulmonary area. Liver, dulness begins at the sixth rib, edge felt one and one-half inch below free margin of ribs. Abdomen is greatly distended, measures thirty-two inches at most prominent part. Many large veins course over it and even extend upward on to the chest. Lower costal margins are prominent and everted. From beneath the left arch emerges a hard mass which fills the whole left side of the abdomen and the right iliac fossa. The mass is hard all over, the surface is smooth, the edges are round. The right side of the abdomen and the left flank are tympanitic. The left lobe of the liver can be felt in the epigastrium, deflected to right of median line. The extremities were slender and poorly developed.

An examination of the blood gave the following result: Red blood-cells, 3,550,000; White blood-cells, 7000.

Differential count of white cells: Lymphocytes, large, 15 per cent., normal, 4–8 per cent.; small, 21 per cent., normal, 35 per cent.; polymorphonuclear, 62 per cent., normal, 62–70 per cent; eosinophiles, 1.4 per cent., normal, ½–4 per cent.

Splenectomy was done on May 17, 1899, by Dr. A. J. McCosh. The patient died three hours after the operation.

Autopsy twenty-four hours post-mortem. Frame, small; adipose, scant; muscle, good. Diaphragm: right side, third rib; left side, fourth rib. Heart: weight, six and one-half ounces. Pericardium, normal; valves, normal. Muscle, anæmic. Lungs: Left; pleura, some loose adhesions; section, tissue firm and tough, looks normal. Right; pleura, adhesions more extensive than on left; section, same as left. Weight, nine and one-half ounces. Peritoneum: Contains 200 c.c. bloody fluid. Mesenteric lymph nodes enlarged, very pale and flabby. Gall-bladder, full of brownish bile. Liver: Consistence, firm; surface, smooth over greater

part, much wrinkled at extreme right; section, chocolate colored; many little white spots like connective tissue, most abundant in wrinkled portion of right lobe. Weight, sixty-eight ounces. Pancreas, normal. Kidneys: Left; adrenal, flattened, but large; ureter, normal; capsule, free; consistence, firm; surface, smooth, flattened against vertebral column; section, straight tubules seem to be generally dilated, so that Malpighian bodies seem to be made up of little cysts; markings of cortex, indistinct. Weight, four ounces. Right, normal in appearance. Weight, five ounces. Stomach, normal. Intestines, normal. Pelvic organs, uterus, infantile. Brain: Tissue very fine; middle commissure, white and tough; two sections made longitudinally through it without severing it. Weight, forty-six ounces. Spleen: This organ was examined both immediately after its removal at the operation and later when hardened in 4 per cent. formalin. Weight, immediately after removal, while still full of blood, twelve and one-half pounds. Total weight of child not more than seventy-five pounds. Form, in general, that of normal spleen. Surfaces: topographically, these correspond to normal spleen, but are, of course, much larger. Upon the external surface are the remains of many fibrous adhesions, which had been divided at the operation. In color, most of the surface is white or yellowish-white, like dense fibrous tissue. Only on the internal aspect and at the postero-inferior portion of the external surface does the color of normal spleen appear. In general the surface is smooth. On the anterior margin there is a deep notch at the junction of the lower with the middle third. Upon section the abnormalities in color are seen to be due to thickening of the fibrous coat, which is in many parts from 2 to 4 mm. in thickness. Where the color of the surface is normal the fibrous coat is normal or only slightly thickened.

The substance of the spleen is unusually firm and resistant. The appearance of the cut section varies in different parts. A central longitudinal section shows numerous very firm white or yellowish-white areas, in general, though very irregularly, of pyramidal form, extending from the surface deeply into the substance of the organ. On such a cut these areas occupy the larger part of the surface exposed. The remainder of the surface has the appearance of normal spleen. These changed areas extend inward about one-half the depth of the organ. Toward the anterior margin nearly the whole section has this appearance, while the normal splenic appearance predominates posteriorly and also about the hilus. In the hardened state the spleen measured from above downward 34.5 c.m., from behind forward 20 c.m., from without inward 11 c.m.

The lymph nodes immediately at the hilus of the spleen are enlarged, soft, and upon section the central parts are deep red, the periphery pale. The enlargement is insignificant in comparison with that of the spleen. Most of the nodes appear somewhat flattened by pressure. Some of the largest measure 2 c.m. in length, 1.25 c.m. in width, and 1 c.m. in thickness.

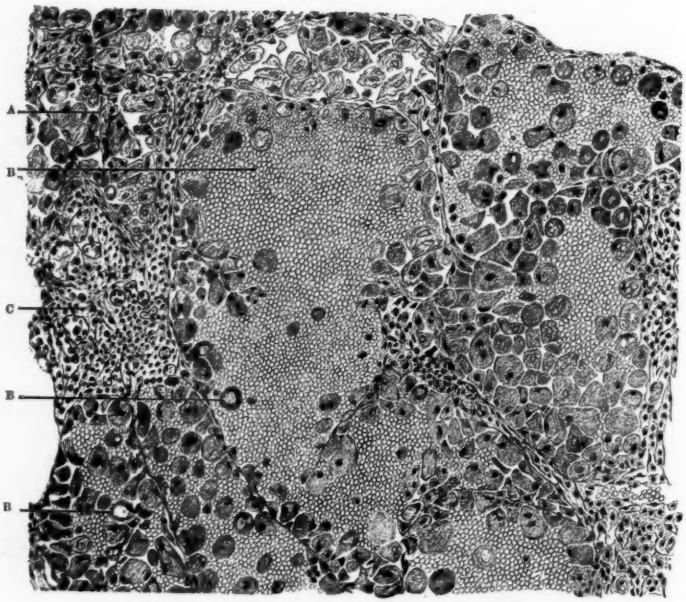
Microscopical Examination. The tissues were hardened in alcohol or formalin and alcohol, embedded in paraffin or celloidin, and sections stained with hæmatoxylin and eosin.

Corresponding with the variations in the gross appearance of the spleen the sections show marked morphological differences. Sections from the hard white areas are very different from those taken from the parts having the appearance of normal splenic tissue. The latter present a

very unusual picture. In its clearest form this consists of large, irregular, round, oval, or polyhedral spaces, more or less filled with large, brightly stained cells. (See Fig. 3.) The size of the spaces varies greatly. The largest measure 0.300 mm. by 0.375 mm.; the smallest, 0.075 mm. by 0.060 mm.; the average seem to be about 0.195 mm. by 0.225 mm.

The walls of these spaces, as seen in cross-section, consist in some parts of a delicate line of connective tissue with infrequent, small, oval nuclei. Frequently adjoining spaces communicate with one another by narrow

FIG. 3.



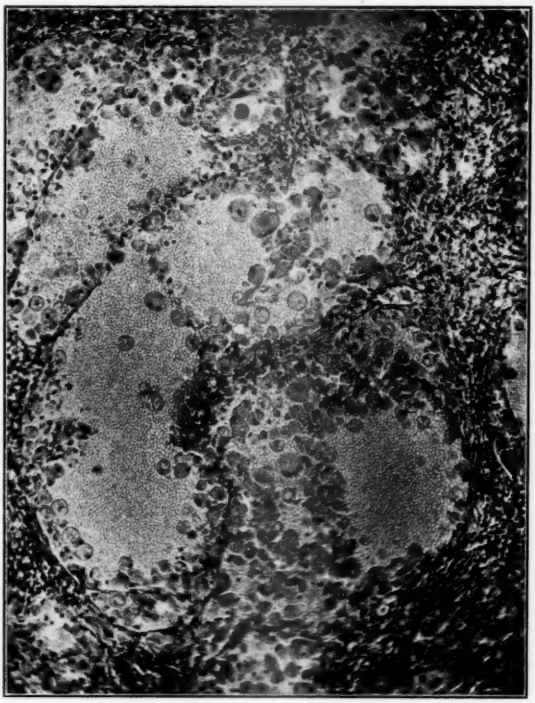
Section of spleen × 800 and reduced.

A. Pulp space filled with endothelial cells. B. Space containing few endothelial cells; centre filled with blood. C. Connective tissue. D. Cells showing pale areas within them.

passages. In most places the walls of the spaces are much thicker. Sometimes the thickened wall consists of simple connective tissue, but where the thickening is most marked the wall is largely made up of a mesh of fine capillaries, the endothelium of which consists of cells having deeply staining round nuclei and a small cell body closely resembling lymphoid cells. The cells within the spaces present a striking appearance. They are very large, as a rule, and of varied shape. Many of them, lying free in the spaces, are round and oval, but where they are closely packed together the form varies greatly. An average cell measures 0.0208 mm. by 0.0260 mm.; the largest, 0.0572 mm. by 0.039 mm.;

the smallest, 0.0130 mm. by 0.0104 mm., little larger than a leucocyte. The nuclei vary considerably in shape and size. As a rule, they are very small in proportion to the size of the cell, measuring 0.0078 mm. by 0 0052 mm., or 0.0130 mm. by 0.0052 mm. Many of the cells show none at all, but from the disproportion between the size of the cells and that of the nuclei it seems probable that, signs of degeneration being absent, in these cases the section had by chance missed the nucleus, though cutting the body of the cell. The nuclei also vary greatly in

FIG. 4.



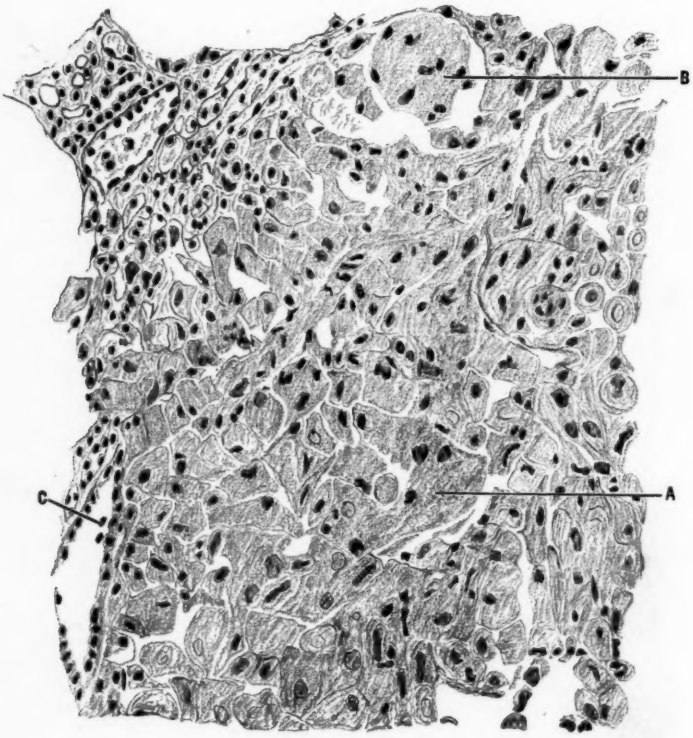
Microphotograph of section of spleen.
Same area as Fig. 1. Magnification about 190.

their staining capacities, some of them staining faintly, some deeply with the hæmatoxylin. Many of the cells show two or three nuclei. Some of the nuclei show mitotic changes, others show nucleoli. The cell bodies also vary in their staining. As a rule, they have taken the eosin deeply, but some stain very faintly. Apart from the staining the appearance of the cell bodies varies. Many of the bodies present a finely granular appearance, some are lightly streaked, while others show one or more round, oval, or irregular areas, which are distinctly lighter and clearer than

the remainder of the cell body, yet are not altogether colorless. In some cases there is only one small area of this kind in a large cell body, in others one or several such areas occupy almost the whole of the cell body.

Many of the cells above described lie free in the spaces. In favorable places, however, it can be plainly seen that many of them are directly attached to or spring from the walls of the spaces in which they lie.

FIG. 5.



Section of spleen × 800 and reduced.

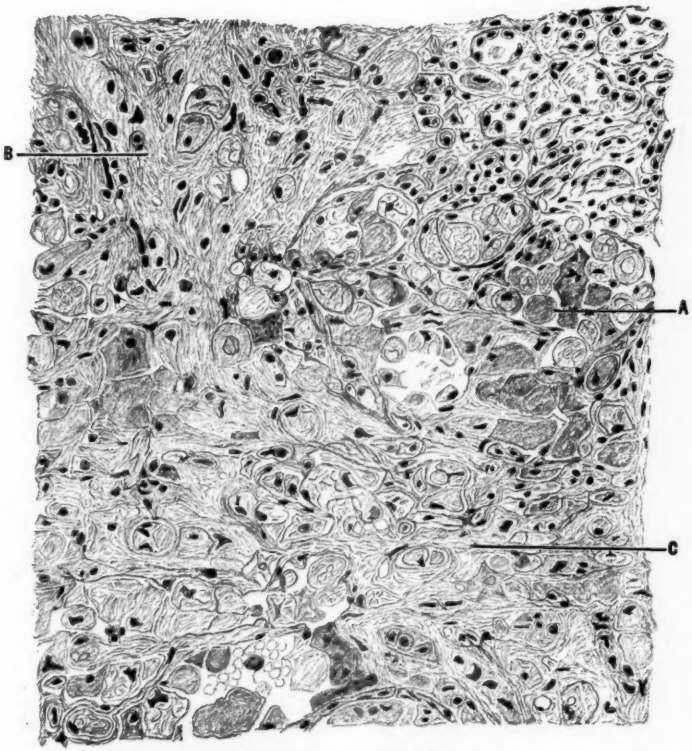
A. Endothelial cells fusing together. B. Giant cell. C. Large vein lined by swollen endothelium.

The number of cells in a space varies in different parts. In some places there are only a few lining the walls or lying free in the centre of the space. In such cases the remainder of the space is filled with blood. In other parts the cells are seen to completely fill the spaces. Careful study leaves no doubt that these spaces are the pulp spaces of the spleen and that the cells that fill them have sprung from their walls. The normal pulp cells have disappeared. On the other hand, Malpighian bodies are met with in the sections almost unchanged. (See Fig. 8.)

From sections showing only partly filled spaces we can pass to others in which these large cells fill and even distend the spaces. In these cases many of the cell bodies apparently fuse together, for we see large, irregular sheets of protoplasm containing many nuclei. (See Fig. 5.) There are also in some places giant cells.

In contrast with these areas, there are parts in which the spaces are apparently lessened in size by the growth of the walls about them.

FIG. 6.



Section of spleen × 800 and reduced

A. Pulp space filled with endothelial cells. B. Delicate connective tissue stroma. C. Area where cells and stroma merge.

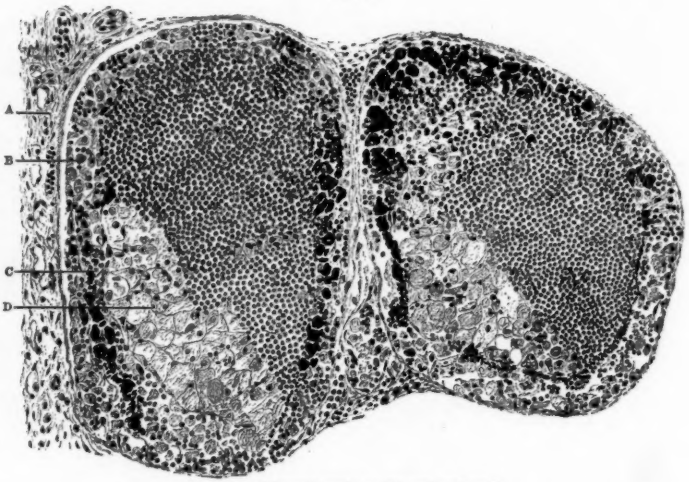
The connective tissue structure in some places is so abundant that the spaces can contain but a single cell. In other parts there are large cells, of the type described above, lying in the midst of a fine fibrillated stroma which closely invests them, and with which the cells mingle in such a way that their bodies can no longer be distinguished from the stroma in which they lie. (See Fig. 6.) In studying these areas one is almost

driven to the conclusion that the large cells are being transformed into connective tissue. Such areas we find on the border zone between the firm white portions and the parts having the appearance of normal spleen.

Sections from the midst of the firm white areas of the organ consist of more or less dense connective tissue. In parts this is infiltrated with small round cells, in other parts it seems to be made up of a meshwork of capillaries, but without the swollen endothelium seen in the walls of the pulp spaces. Many of these areas have the appearance of œdematous connective tissue.

The capsule of the spleen and the trabeculæ are greatly thickened. The bloodvessels are regularly surrounded by a greatly increased quantity of connective tissue. The Malpighian bodies are not much altered.

FIG. 7.



Section of lymph node × 800 and reduced.

A. Thickened capsule. B. Endothelial cells in lymph-sinus. C. Pigment in lymph-sinus. D. Delicate reticulum, spaces filled with endothelial cells.

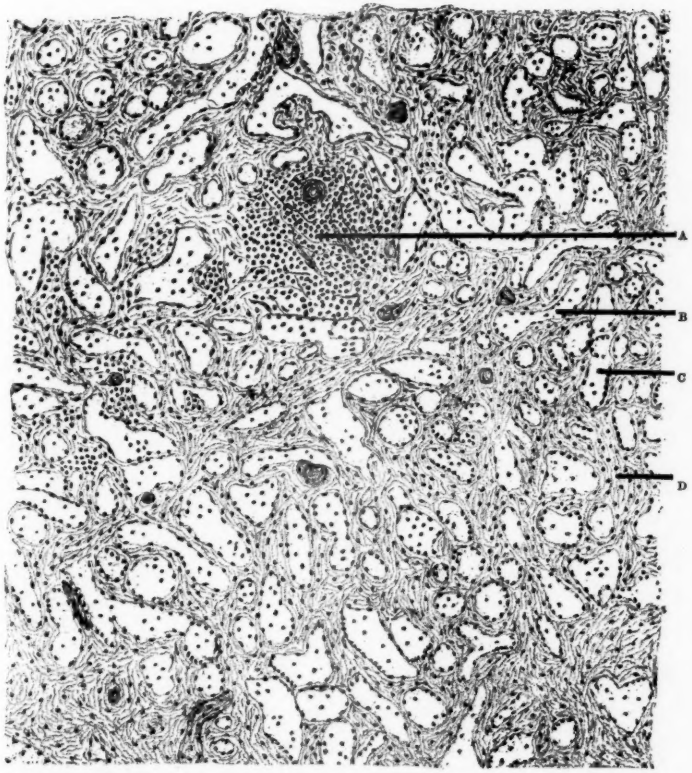
In some places the changes in the pulp seem to encroach upon the bodies. Sometimes the periphery of one of the bodies contains an unusual number of small capillaries. In general, however, they seem very little affected by the great changes that have gone on about them. Sections of the spleen were stained with methylene-blue and carefully examined for bacteria, but none were found.

The lymph nodes immediately in relation to the spleen show very interesting changes. In the gross they are swollen, but rather soft. On section the centre of the node is deep red, the periphery pale; sections present a beautiful microscopic picture. (See Fig. 7.) Each follicle is outlined by a wall of brightly shining, rather dark pigment, which closer inspection shows to be deposited in the inner part of the lymph-sinus surrounding the medullary portion of the follicle. In some places

the pigment quite fills the sinus. The wall of pigment is almost continuous about the follicle. The part of the lymph-sinus not occupied by pigment is seen to be nearly completely filled by cells. A few of these are normal lymphoid cells. The greater number are large round, spindle-shaped, or polygonal cells with large round or oval nuclei.

Both the nuclei and the cell bodies stain rather faintly. In parts where the number of these cells is small they may be seen to spring

FIG. 8.



Section of spleen from a case of chronic empyema. Simple dilatation of cavernous veins or pulp spaces. × 480.

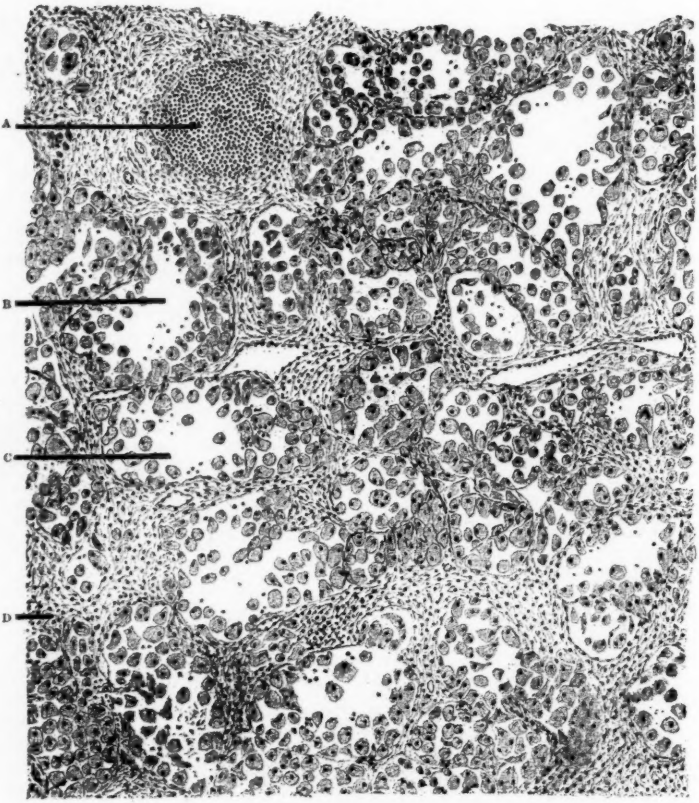
A. Malpighian body. B, C. Dilated spaces. D. Connective tissue septa.

from the walls of the reticular tissue of the lymph-sinus. When there are many of these cells packed together this relation is obscured.

Throughout the node the medullary portion of the follicle has undergone remarkable changes. From a greater or less part of the medulla, usually a segment of the periphery, the lymph cells have almost com-

pletely disappeared. In the transformed areas the reticular substructure is plainly seen, with here and there one of the branching cells of which it is made up. Within the irregular spaces of this network lie large cells, one cell usually filling a compartment, though in some cases several may be seen in one space. These cells stain very faintly. They are round or polygonal. The nuclei are of fair size, round, oval, or irregular, and stain feebly.

FIG. 9.



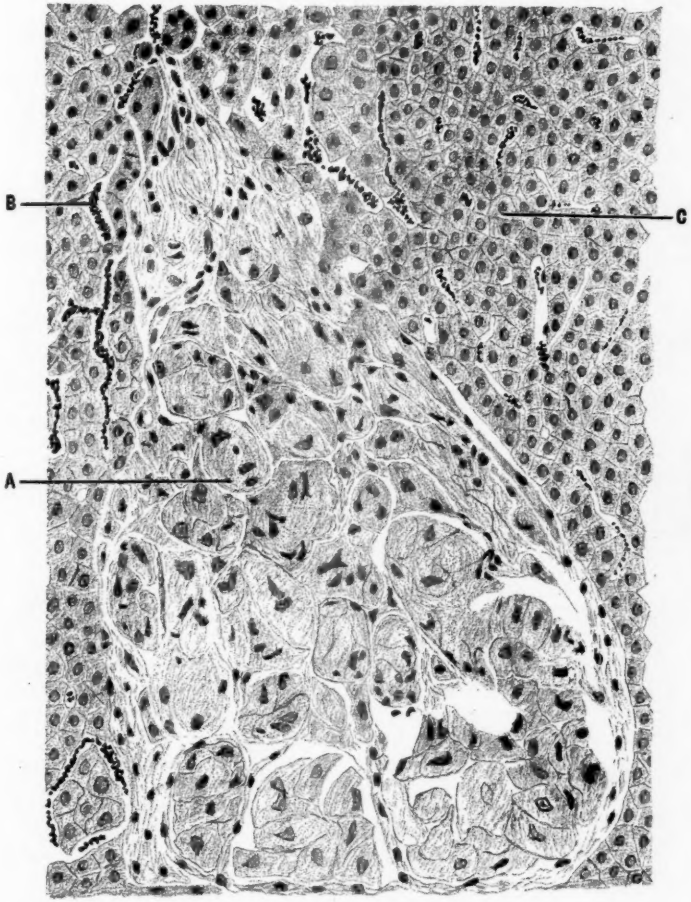
Section of spleen. Splenomegaly. × 480.

A. Malpighian body. B, C. Dilated spaces filled with endothelial cells. D. Connective tissue septa.

Some of the cells show paler areas within them just as described in the cells of the spleen. The whole follicle may have undergone these changes. Usually a part of the follicle presents a normal appearance. The transition from the changed almost colorless zone to the darker part filled by the lymphoid cells is abrupt. The lymphoid cells are nor-

mal in form and arrangement. Here and there through them a large light-colored cell may appear, or the normal reticulum may be seen. A few pigment granules may be scattered through the medulla of the

FIG. 10.



Section of liver × 800 and reduced.

A. Endothelial cells filling spaces of connective tissue. B. Pigment in capillaries. C. Normal liver cells.

follicle, either in the changed or normal areas. The smaller arteries or capillaries occasionally appear in the sections, when it can be seen that they are lined by a swollen endothelium which, in parts at least, shows

a fine granular pigmentation. The capsule of the node is thickened and filled with small bloodvessels, many of which are lined by unusually large and prominent cells. The connective tissue surrounding the follicles is similarly changed.

The cortical portion of the mesenteric nodes is practically normal. The capsule is not thickened and there is no thickening of the trabecula. The medullary part of the node, on the other hand, presents changes exactly similar to those described in the medulla of the follicles of the nodes found near the spleen—that is, the lymphoid cells have almost disappeared, the normal reticulum stands out prominently, its spaces being filled with the large, irregular, almost colorless cells above described. These cells in the mesenteric nodes stain even more faintly than those in the nodes at the hilus of the spleen. The affected area is so pale that even to the naked eye the demarcation between the cortex and medulla of the node can be plainly seen. Pigment is present, but it occurs only in scattered granules. There is none of the massing of pigment in the lymph sinuses which marked the other nodes.

The liver also presents changes of interest. The capsule is thickened and the connective tissue throughout the organ is increased. The capillaries everywhere contain considerable pigment like that seen in the lymph nodes. The liver cells are normal. The bile ducts are normal. In the blood that fills the branches of the portal vein there are large cells of the type seen in the pulp spaces of the spleen. Considering the number of these cells free in the pulp spaces of the spleen and the fact that these spaces are the radicles of the splenic veins, this is not surprising. The walls of the veins containing these cells are normal.

In none of the many sections examined was there found any evidence that these cells had effected a permanent lodgement within or grown from the branches of the vein in which they were present. However, in many of the areas of perilobular connective tissue an arrangement or development extremely like that described above as seen in the lymph nodes can be seen. (See Fig. 10.) This consists of irregularly round or oval spaces bounded by delicate connective tissue walls, and filled with large, pale cells of irregular shape, and containing large nuclei. The cells are remarkable for their large size, both of body and nucleus. In many of the spaces the cells fuse together so completely as to make it very difficult to distinguish the outlines of the individual cells.

When first seen these areas were looked upon as almost certainly metastases. Further consideration has led the writer to the opinion that these cells have been developed from the tissue in which they lie. The areas in which these cells occur are in all cases small, although from the clinical history the process in the spleen must have been going on for thirteen years. The growth of cells, while it distends or enlarges the size of the connective tissue areas in which it occurs, never oversteps the limits of the connective tissue or advances into the surrounding tissue. The integrity of the branches of the portal vein has already been mentioned.

With these facts in mind, it seems fair to conclude that these cells in the liver are not metastases, but have been developed in the situations in which we find them from the endothelium lining the lymph spaces of the connective tissue.

The lungs show a slight thickening of the walls of some and a dilatation of others of the air spaces. There is congestion of all the vessels, and in the perivascular lymph spaces surrounding the larger vessels there is a moderate amount of dark, almost black pigment.

The heart is normal.

The kidney: The left shows a remarkable dilatation of several of the straight tubules by which the surrounding tubules are much compressed. In some of the sections there are small areas of lymphoid infiltration. Otherwise the organ is normal. There is no pigment present. The suprarenal shows some degeneration of the cells of the medullary portion, but in the main is normal.

The intestine in some parts shows a moderate degree of simple hypertrophy of the lymph nodules, otherwise is normal.

The pigment observed in the lymph nodes and liver deserves some further description. In fine granules it has a light yellow hue, in masses it is dark brown. In the lymph nodes some of the granules are evidently intracellular, some of them, especially in the lymph sinus, are free. The relation of the larger masses to the cells cannot be made out. In the liver the pigment is found free in the capillaries, not at all in the cells. Both in the nodes and in the liver the pigment in sections treated with solutions of ferrocyanide of potassium and hydrochloric acid gives the iron reaction. Its distribution, the lymph sinuses of the lymph nodes and the capillaries of the liver show that it must have come from the spleen. Such pigment formation in enlargement of the spleen is not unusual. In this case the amount of pigment is remarkable. The pigment above described corresponds to the hæmosiderin of Neumann.

The pigment observed in the lymph spaces surrounding the vessels of the lung is black in color and does not give the iron reaction. It is, therefore, not of the same origin as that in the lymph nodes or liver.

When, with these descriptions of these cases and the results of the morphological examination before us, we endeavor to name the lesion we find ourselves facing a difficult problem. At first sight it seems that we have to do with an endothelioma of the spleen, with metastases in the lymph nodes and liver. The huge spleen is made up in part of areas of connective tissue, in part of areas in which the pulp spaces of the spleen are filled with endothelial cells. The normal cells of the splenic pulp have disappeared. Under the microscope there is nothing but the shape and arrangement of the spaces in which these endothelial cells lie and the presence of the Malpighian bodies to show that it is the spleen we are dealing with. In the lymph nodes we find a similar process, the disappearance of the normal cells of the organ and the development of an unusual number of endothelial cells. In the liver, also, we find areas in the perilobular connective tissue in which there has been an unusual development of endothelial cells in the lymph spaces. It must also be noted that the arrangement of these cells in the connective tissue of the liver corresponds to some extent to that found in the endotheliomas of the pleura described by many observers. Against the acceptance of this hypothesis we have the following considerations: First of all it is to

be remembered that the process, whatever its nature, had existed for thirteen years in the case that came to autopsy, and that even then the condition of the patient was not incompatible with the prolongation of life for some years further. Moreover, the same process, in all probability, is developing in a younger sister at exactly the time of life the disease appeared in the elder. These facts unquestionably point to the action of some systemic poison rather than to a tumor.

With regard to the spleen itself, we note a number of facts not consistent with the hypothesis of endothelioma. After all the years the process has been going on the organ is still a spleen—the shape (the surfaces are those of a spleen) and even the notch in the anterior border are preserved. Moreover, while the spleen was adherent there was no direct extension of the process beyond the bounds set by the capsule of the organ. Within the spleen itself we find also that the spaces in which the endothelial cells lie are those which normally belong to the spleen, the walls of the spaces being the connective tissue framework of the spleen itself. This point can be well brought out by contrasting with a section of the spleen in question another taken from a case of empyema, which is characterized by simple dilatation of the pulp spaces of the spleen without the endothelial multiplication. (See Figs. 5 and 6.) In this case we find the round or oval spaces separated by walls of delicate connective tissue lined with small endothelial cells. In the spleen we are studying we find like spaces filled with cells, which we have shown have sprung from the endothelium lining their walls. The essential difference between the two is the size and number of the endothelial cells in the latter case. If increase in size and number of endothelial cells constitutes an endothelioma, then this process must be so classified; but by a similar line of argument any simple hyperplasia of the spleen marked by multiplication of the proper cells of the splenic pulp would become a tumor of the spleen. Finally, the decisive objection to the endothelioma hypothesis rests in the fact that there is nothing that can be regarded as an autonomous growth within this spleen. Neither on gross nor microscopic examination can we define a growth independent of the structure of the spleen itself; in fact, as has been pointed out, the very opposite is true.

The requirement that a tumor must be an autonomous growth, enunciated as a doctrine by Thoma,[14] is indorsed by Ziegler,[15] Lubarsch,[16] Prudden[17] and others. If we accept that doctrine we cannot regard the change in this spleen as a tumor, and it cannot, therefore, be called an endothelioma. We are driven to regard the process as a hyperplasia of the spleen characterized by an unusual development of endothelial cells and the transformation of a considerable part of the organ into dense connective tissue. It is possible in some of the sections to demonstrate, so far as such a process is demonstrable, that some of the endothelial cells go to form part of the connective tissue increase.

The question next arises what relation, if any, the changes in the lymph nodes and liver bear to the process in the spleen. Are they metastases or not? There are good reasons for a negative reply.

With respect to the lymph nodes, the proliferation of endothelial cells both in the lymph sinus and in the medulla of the node is exactly similar to that frequently seen in other diseases. It is to be noted that practically every follicle of a node is similarly affected, and yet in almost every one of them some part of the follicle remains normal. The delicate reticulum of the node is everywhere preserved. There is not the unruly growth characteristic of malignant metastases.

The condition found in the liver, while, as already admitted, similar to that described as endothelioma of the pleura, etc., is a diffuse, not a localized process. It occurs in all parts of the organ. The individual areas are very small. The walls of the hepatic artery and portal vein are not affected. The cells brought from the spleen, many of which can be seen lying within the branches of the portal vein, have not been able to effect a permanent lodgement within them, and as there is no direct connection of the lymph spaces of the liver with those of the spleen, the cells cannot have been lodged in the liver by that channel. Finally, the increase in the endothelial cells is limited wholly to the connective tissue areas, and in no part have the cells spread beyond the limits of these areas. The liver cells proper are unaffected by the process. The writer, therefore, regards it as reasonable to look upon this multiplication of endothelial cells in the lymph spaces of the connective tissue of the liver as not the result of metastases.

Viewed as a whole the characteristic feature of the process in spleen, lymph node, and liver is the multiplication of endothelial cells, associated in the case of both spleen and liver with a great increase in the connective tissue of the organ.

If, as the writer believes, the processes in the lymph nodes and liver are not metastases, we must suppose that some influence has affected these several organs in the same way. The process in the spleen and lymph nodes is exactly similar, though much greater in degree, to the changes that are sometimes observed in typhoid fever, tuberculosis, and other infectious diseases.

Literature dealing with similar cases is extremely scanty. In a *Thèse de Doctorat*, by E. Gaucher, Paris, 1882, the writer is persuaded that the same affection is described under the title of "Splenomegalie Primitive—Epithelioma primitif de la rate." An epitome of the thesis runs as follows:

The patient was a woman who died at the age of thirty-two years from an intercurrent pulmonary tuberculosis. The splenic enlargement dated from the age of seven years; had therefore existed for a period of twenty-five years. It had been accompanied by repeated epistaxis, ulceration

of the gums and oozing of blood therefrom, occasional attacks of abdominal pain, disturbance of the functions of the stomach and intestines, and occasional jaundice. None of these attendant symptoms were constant. The blood showed the changes of simple anæmia. The patient finally died of exhaustion due to tuberculosis. The autopsy showed tubercular lesions of the lungs and peritoneum, moderate enlargement of liver, a huge spleen weighing 4 kilogrammes 770 grammes, normal lymph nodes. The enlargement of the liver was found to be due to a diffuse cirrhosis which the author considered secondary to the splenic enlargement. The spleen, both macroscopically and microscopically, presented lesions in all essential respects the same as those in the writer's case. The drawings which accompany the thesis leave no doubt of this. The nature of the error by which the designation of primary epithelioma of the spleen is given is made clear by the author's remarks. He calls the large cells found in the spleen epithelial or epithelioid by reason of their morphological appearances and their staining relations, and does not classify them according to their origin.

In Bouchard's *Pathological Anatomy*, 1899, vol. iii., part ii., p. 894, a cut of Gaucher's is reproduced under the designation of endothelioma of the spleen.

Following Gaucher, Picòu and Raymond[1] present a report of a spleen removed by operation from a woman of thirty-two years. The enlarged spleen was mistaken for a uterine fibroid, because of a history of menorrhagia for a year before the operation. The only other symptoms were vague abdominal pains, extending into the lower extremities, swollen and bleeding gums, some disturbance of digestion, and œdema of the ankles. The patient recovered from the operation. The spleen presented the lesions described by Gaucher, and the reporters accepted his title of primary epithelioma. The blood showed the changes of simple anæmia. In the report of Picòu and Raymond occurs this significant paragraph: "In the opinion of Cornil[26] the lesions described by Gaucher cannot be primary epithelioma. This author does not admit the existence of primary epithelioma of the spleen. Despite the appearance, he believes this case to be a primary hypertrophy of the spleen, with proliferation of the reticular tissue. As for the cells, they recall the appearances he has often observed in glandular hypertrophies."

In English literature William Collier[2] presents a detailed account of the lesions found in a girl, aged six years, whose death is said to have been accelerated by an attack of "epistaxis and sickness." An elder sister of the case reported had also died with a greatly enlarged spleen. The lesions of the spleen found by Collier correspond to those in the writer's case. The lymph nodes were also enlarged, and presented lesions similar to those described by the writer. Collier is evidently

inclined to call the process endothelioma. The specimens were submitted to the Morbid Growths Committee of the London Pathological Society, who call attention to the similar affection in the sister, note that the most striking feature in the microscopical examination of sections from the central part of the spleen is the presence of an enormous number of endothelial-like cells, but add that "this appearance is similar to that which is not uncommon in the lymphatic glands, and was present in the lymphatic glands in this case." The committee's position is evidently that of mild dissent from the author's view. The blood examinations made by Collier showed that the case was not one of leukæmia.

Weichselbaum,[3] in Germany, reports the examination of a spleen removed from a soldier, twenty-one years of age, under the title "Primary Multiple Endothelial Sarcoma of the Spleen." No history is given. The microscopical lesions described are strikingly like those described by the writer in the parts of the spleen shown in Figs. 5 and 6. Weichselbaum calls attention to the apparent fusion of the endothelial cells with the reticulum of fibrous tissue in which they lie. So high an authority as Birch-Hirschfeld[4] has reviewed Weichselbaum's case, and declares that the new formations were "not tumors, but a large-celled focal hyperplasia."

The writer may therefore claim the authority of Cornil, the Morbid Growths Committee of the London Pathological Society, and Birch-Hirschfeld for the view that he has taken of the lesions found in these cases.

The writer's cases are of almost as much interest clinically as pathologically. Their clinical relations may be briefly summarized thus:

Case I. Great enlargement of both liver and spleen, simple anæmia, slight enlargement of superficial lymph nodes.

Case II. Enormous splenic enlargement and moderate enlargement of the liver, simple anæmia, slight enlargement of superficial lymph nodes; affection of thirteen years' duration. Patient's sister had enlargement of spleen beginning at practically the same age. It may therefore be safely assumed that whatever the disease it is the same in the two cases.

From both the history of the patients and the clinical observations it is apparent that the enlargement of the liver is secondary to that of the spleen.

So far as the enlargement of the superficial lymph nodes is concerned, it being so insignificant in comparison with the enlargement of the spleen, and, furthermore, an equal enlargement of the superficial nodes is so commonly met with in children whose nutrition is under par from any cause, that it seems that this feature of the cases may be safely dropped from consideration. We have, therefore, left for study only the splenic and hepatic enlargements with the blood changes of simple anæmia.

While simple anæmia may be accompanied by moderate splenic enlargement, no one would suggest that the anæmia in these cases could account for the splenic enlargements. Either the enlargement of the spleen is the cause of the anæmia, or they are dependent upon a common cause, or have no relation to one another. At all events, the explanation of the progressive enlargement of the spleen becomes the important point in these cases.

Chronic enlargement of the spleen may be due to various influences, such as cardiac disease, or any cause of continued enlargement in the portal system, amyloid degeneration of the viscera, the blood diseases, leukæmia, etc., tuberculosis, rhachitis, malaria and syphilis. (The rarity of primary new growth of the spleen, the length of time the disease had existed in the first case, the occurrence of the affection in a sister at the same period of life, were, in the early study of the cases, held sufficient grounds for the exclusion of the hypothesis of a new growth.) The clinical history, the absence of temperature, the absence of any evidence of tuberculosis elsewhere excluded tuberculosis. No cause for venous enlargement or amyloid degeneration could be found. The blood examinations excluded leukæmia. As for rhachitis, the splenic enlargement only began at the age of three years, while that of rhachitis occurs during the first or second year; moreover, there is no history of rhachitis, and the children showed no evidence of any previous rhachitic affection.

The absence of malarial history, the negative results of examinations of the blood for malarial organisms, the moderate degree of anæmia in comparison with the size of the splenic tumor, and finally the impotence of arsenic, seem to fairly exclude the possibility of progressive malarial poisoning.

Hereditary syphilis presents the final and most difficult alternative. The history of the death of four children during infancy, together with one still-birth, certainly suggests the possibility of specific disease in the family, although the early deaths were attributed to "summer complaint" and were not consecutive. The weight of evidence is, however, altogether against the acceptance of syphilis as the explanation of our problem. So far as could be learned the children that died gave no evidence of syphilis. The mother herself bears no traces of such disease. Three other living children, intervening in age between the two sisters, are perfectly healthy; and, finally, apart from the splenic enlargement, neither of our patients presents any of the characters of hereditary syphilis. In the face of such facts it would not appear reasonable to maintain that we are here dealing with hereditary syphilis, finding its only expression in the enlargement of the spleen and liver. It being therefore impossible to account for the splenic enlargement upon the basis of any of the affections enumerated, we are led to conclude that we have to do with a primary or idiopathic enlargement of the spleen.

The changes in the spleen (and liver) are accompanied by simple anæmia.

The ages of the patients, as well as the absence of leucocytes or any morphological changes in the red cells, show at once that we are not dealing with the affection peculiar to infancy, described by von Jaksch,[5] Loos,[6] and Luzet[7] as anæmia infantum pseudoleukæmica.

The cases do present a close relationship in their clinical features to the affection variously described as splenic anæmia or splenic pseudoleukæmia. The use of the latter term seems only to increase the confusion of a sufficiently difficult subject, and is avoided. The subject of splenic anæmia has been thoroughly discussed lately by Sippy[27] and Osler.[28] It is sufficient for our purposes to note that its characteristic features are considerable enlargement of the spleen, associated with more or less marked anæmia. The pathology, so far as known, is that of chronic interstitial splenitis.

From the type of splenic anæmia the cases herein reported present some points of difference.

1. The age of the patients, or, better, the age of the patients at the beginning of the splenic enlargement. Splenic anæmia is essentially a disease of adult life. Of late, Carr,[18] Fox and Ball[19] in England and Glockner[20] and others in Germany have reported a number of cases of splenic anæmia in children, most of them under two years of age, and only one traced to the age of three and a half years. The condition is evidently the simple anæmia associated with enlargement of spleen and possibly liver, due, as a rule, to rickets or syphilis. During this early period enlargement of the liver is so common from a fatty infiltration that that symptom should be given much less weight than belongs to it at a later age. From the reports cited we learn that splenic anæmia regularly yields to dietetic treatment with cod-liver oil and iron, yet, from time to time, cases are seen in which the tendency of the spleen to hypertrophy cannot be controlled by any form of treatment. None of the latter cases have been followed to the end. In all the autopsies recorded the lesion of the spleen was a chronic interstitial inflammation with hypertrophy. The writer's cases belong neither to the adult nor to the infantile type of splenic anæmia, but stand between them.

2. The progressive character of the malady. The enlargement of the spleen and the anæmia advance despite all forms of treatment. Splenic anæmia is regularly amenable to treatment.

3. Despite its progressive character, the affection seems not to be fatal *per se*. Gaucher's case lived to her thirty-second year and finally succumbed to tuberculosis. The first of the writer's patients reached the age of sixteen, and would probably have lived some years longer without operation. Although the second case is developing some cachectic symptoms there is no prospect of early fatal conclusion to the process.

4. The chronicity of the affection. The enlargement of the spleen in Gaucher's case dated back twenty-five years, in the elder of the writer's cases thirteen years.

5. The enlargement of the liver. This does not belong to the adult type of splenic anæmia, and its occurrence in the infantile type, as above suggested, is probably due to the fatty infiltration common at that age.

6. The huge size attained by the spleen, transcending all other features of the affection, and rarely seen in any other affections except chronic malaria or leukæmia.

7. The occurrence of two cases in sisters in the report of Collier and the same phenomenon in the writer's cases is worthy of note. This feature of the cases certainly suggests the action of some systemic poison affecting several members of a family.

8. The cases of Gaucher, Picòu and Raymond, Collier and the writer are all females.

The only affection that one can suggest which meets the requirements of these cases is hereditary syphilis, yet, as already seen, there is no other evidence of the presence of this disease in the family.

It cannot be assumed that the points enumerated will suffice for a complete differentiation of our cases from splenic anæmia. In France, the terms splenomegaly and splenic anæmia have evidently been used as synonymous. Debove,[21] Bruhl,[22] Peter,[23] Rendu[24] and others have reported cases of adult splenic anæmia as splenomegaly, and Gaucher[25] has maintained that the cases in question belonged to the type he had described in his thesis.

In Italian literature we find still another designation for these cases. The first reports came from Banti,[9] under the title "Splenomegalia primitiva con cirrosi epatica," which has been followed by Rinaldi,[10] Bonardi,[11] Maragliano,[12] Casarini[13] and others. Sometimes it is called malattia del Banti. Banti himself admits the close relationship of the affection he has described with splenic anæmia and the possibility that it is identical. Among the cases in Italian literature are some, notably one of Rinaldi's, presenting very close resemblance to the writer's cases. At present the only sufficient distinction of the type of disease herein reported is the pathology. That is altogether different from anything else to be found in medical literature.

Upon the basis of the observations recorded above the writer believes that it can be maintained:

1. That the affection first described by Gaucher as primary splenomegaly, or primary epithelioma of the spleen, is a definite and distinct disease.

2. That the process is not a new growth, but an endothelial hyperplasia of the spleen, and that it may be associated with like changes in the retroperitoneal and mesenteric lymph nodes and the connective tissue of the liver.

The symptomatology of the affection as derived from Gaucher's thesis and the writer's cases may be briefly summarized thus:

1. Enlargement of the spleen, beginning in childhood (second to seventh year), slow, progressive.

2. Enlargement of the liver, secondary to that of the spleen, may be considerable, but never reaches the extent of the splenic affection.

3. Simple anæmia. The only changes observed in the blood are those associated with any chronic enlargement of the spleen.

4. Softening of the gums with oozing of blood.

5. Epistaxis repeated. Osler has noted the association of this symptom with chronic splenic enlargements.

6. Cutaneous hemorrhages and icterus, present in Gaucher's case, not in the writer's.

7. Symptoms referable to the mechanical effect of the splenic enlargement: Pain in the abdomen; disturbance of functions of stomach and bowels; dyspnœa; dysuria; cramps in the legs.

8. The case recorded by Picòu and Raymond shows that the earlier stages of the disease may be overlooked and the first evidence of the enlargement of the spleen be the effect of its weight.

9. The problem of the clinical differentiation of cases of the type herein reported from splenic anæmia must be left to the experience of the future.

In conclusion, the writer must acknowledge his indebtedness to the attending staff of the Presbyterian Hospital for the many courtesies extended in allowing him to follow the cases while in that institution and to make use of their records. He is especially indebted to Dr. G. A. Tuttle for the record of the autopsy and the material from which his studies were made. To Dr. Prudden, the Director of the Pathological Laboratory of the College of Physicians and Surgeons, the writer would express his most sincere thanks for the criticism and counsel which guided him all through his work.

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PRIMARY ECHINOCOCCUS CYSTS OF THE PLEURA.

REPORT OF A CASE OF PRIMARY EXOGENOUS ECHINOCOCCUS CYSTS OF THE PLEURA, SHOWING HYALINE DEGENERATION OF THE CUTICLE WITHOUT LAMELLATION, WITH NOTES FROM THE LITERATURE.[1]

BY CHARLES CARY, M.D.,
PROFESSOR OF CLINICAL MEDICINE, UNIVERSITY OF BUFFALO, N. Y.,
AND
IRVING P. LYON, M.D.,
INSTRUCTOR IN CLINICAL MEDICINE, UNIVERSITY OF BUFFALO, N. Y.

IT has seemed advisable to report in detail the following case of echinococcus cysts of the pleura, both because of the rarity of hydatid disease in America, and especially because of the atypical degeneration of the cyst-walls found in the case, obscuring their true nature and causing difficulty in the diagnosis.

Summary. Physical signs of chronic pleurisy, with adhesions and effusion at right base; aspiration of albuminous fluid loaded with cholesterin crystals; clinical diagnosis of chronic encysted pleural effusion. Operation. Lung free, no adhesions, no effusion; removal from pleural cavity of a small mass of collapsed cysts resembling grossly echinococcus cysts. Microscopical

[1] Read before the Association of American Physicians, at Washington, D. C., at the fifteenth annual session, May 3, 1900.

examination. Outer cyst-wall thin, hyaline, without lamellation, and infiltrated with cholesterin crystals; lining membrane degenerated and fatty, and containing much cholesterin, but no scolices or calcareous corpuscles; final discovery, after continued search, of two echinococcus hooklets. Anatomical diagnosis: Degenerated, exogenous, primary echinococcus cysts of pleura.—J. O., aged thirty-one years, a mulatto (mother black, father white), single, a kitchen servant in restaurants, applied for treatment at the dispensary of the University of Buffalo, January 26, 1900, complaining of shortness of breath on exertion, dull pain in the lower right chest, and inflammation of the bladder. The diagnosis of chronic pleurisy, with adhesions and effusion, was made, and the patient was admitted to the Buffalo General Hospital, February 2, 1900.

Personal History. The patient was born in Virginia and lived in that State until the age of twenty-one, and *had never been outside the United States.* He had malaria, at Newport News, at the age of twenty-one, and typhoid fever, at Charlottesville, at twenty-three. About two years ago he had pneumonia, and was in the hospital at Charlottesville from January to April, 1898. In convalescence he developed pleurisy on the right side and was tapped of "about three quarts" of a clear yellow fluid; recovery, except for a "tender feeling" in the lower right chest occasionally on catching cold. In the autumn of 1898, at Washington, D. C., he developed pleurisy on the right side, and "about three quarts of a yellowish fluid containing some white matter" was aspirated from the chest. After this he remained well until the autumn of 1899, when a dull aching pain developed in the lower right chest, with dyspnœa on exertion, requiring him to stop work at about Christmas. About this time, also, he first noticed that his urine contained considerable white sediment and that his micturition was frequent and was accompanied with pain at the end of the act, which was referred to the glans penis. He denied any history of gonorrhœa, but admitted having had a sore, at the age of twenty-five years, on the penis, which was followed by a blotchy eruption over the body and general bone-aching. He took treatment, and recovered. Since his present illness began, about last Christmas, he has lost considerable weight and strength, and has been unable to work. He has never had jaundice to his knowledge. In regard to his association with dogs, he said that he always lived with them until he left home at the age of twenty-one, since which he has not had much to do with them.

Condition on Admission. The patient was a small, thin, anæmic, sickly-looking mulatto, weighing 115 pounds. A blotchy pigmentation was observed over both cheeks. Lips and mucous membranes were pale, conjunctivæ clear; finger tips and nails showed no clubbing or incurvation. Temperature, 98.5° F.; pulse, 78; respiration, 24.

Thorax. On *inspection,* nothing distinctive noted; no bulging; respirations shallow and increased in frequency. On *palpation,* over the lower right chest in the axillary line at the end of deep inspiration was felt a very rough, prolonged, vibrating friction of maximum intensity. Vocal fremitus was diminished over the right base up to the level of the fifth space; above this normal. On *percussion* there was marked dulness over the right base, rising to the level of the fifth space in the axillary line and sagging somewhat below this level in front and dropping abruptly behind close to the spinal column. The percussion note was not quite flat over the base, though nearly so in the midaxillary line.

The upper limit of dulness moved very slightly on change of position. On *auscultation*, over the area of dulness the breath sounds were not audible, but the spoken voice could be heard faintly. At the end of inspiration a rough, harsh, loud friction sound was heard in the axillary line as high as the fifth space. No râles were heard. The thorax otherwise showed nothing abnormal.

Heart. Point of maximum impulse in the mammillary line in the fifth space ; sounds clear.

Liver. Edge could be felt just at the costal margin.

Spleen. Not palpable.

Abdomen. Negative; no tenderness on pressure over flanks or hypogastrium.

Genitalia. Endoscopic examination (kindly made by Dr. E. J. Meyer) showed a stricture of the membranous urethra, and superficial ulceration of the prostatic urethra; no growths or cysts in the bladder.

Urine. Sp. gr., 1017, slightly alkaline, considerable albumin, no sugar, considerable pus, and an occasional hyaline cast.

Sputum. [No material from the deeper air passages could at any time be obtained.]

The *diagnosis* of chronic pleurisy with adhesions and effusion was made, and on February 10th exploratory puncture with a hypodermatic needle was made. *The needle was inserted four times without result in different places over the area of dulness, but on the fifth puncture, in the sixth space in the anterior axillary line, the syringe filled with fluid,* which was of a straw-yellow color, and opalescent on agitation. It was found to contain a large number of cholesterin crystals, a considerable amount of albumin, and a trace of sugar. Tubercle bacilli were not found.

The large amount of cholesterin crystals in the fluid excited suspicion of old cystic formation, but as no evidence of echinococcus was found the case was assumed to be one of simple, chronic, *encysted,* pleural effusion. After a few days, as no improvement had taken place, it was decided to withdraw the fluid by aspiration. On this occasion *the needle was inserted also five times before fluid was obtained,* when about one-half litre of fluid of the same character as above described was withdrawn.

A week later dulness was still found over the right base, though less marked than before. Dr. Roswell Park saw the case at this time and suggested the possibility of hydatid cyst. The patient remained unimproved, and it was decided to open the supposed encysted cavity and produce its obliteration.

Operation. Operation was performed by Dr. Roswell Park, on February 24th. Before incising the chest-wall a small syringeful of fluid was aspirated from the right base on the first puncture. This fluid was in every way similar to the fluid previously aspirated. The chest-wall was then opened and several ribs were resected. The costal pleura was found considerably thickened. After entering the pleural cavity, to our surprise, the lung was seen to be free from adhesions, and no encysted cavity or fluid was found. The lung showed on its surface several small, round, opaque, nodular spots that suggested calcified tubercles. No fluid could be found, and only about two ounces of bloody fluid could be drained from the chest by turning the patient on the right side. In exploring the pleural cavity with the hand *a soft mass was withdrawn without appreciable resistance,* which was about the

size of a lemon and consisted of a mass of collapsed cysts closely united together. No fistula through the diaphragm could be felt. The operation was terminated by placing drainage in the chest cavity.

Subsequent History. The patient rallied well from the primary operation, but his pulse, temperature and respirations tended to increase with considerable irregularity. About one week later his condition grew worse and a secondary operation was done, enlarging the opening in the chest and cleansing the cavity, which had contracted down to rather small size. Free drainage and irrigation of the cavity were maintained throughout the course of the case. The general condition of the patient tended gradually downward with high and irregular rate of pulse, temperature, and respiration. During the last week of illness the temperature ranged irregularly between 99° and 104° F., the pulse ran from 120 to 140, and the respirations were from 40 to 52 per minute. The physical signs of pneumonia were not found. The patient died on May 4, 1900, without the development of special symptoms. Autopsy could not be obtained.

Pathological Notes on the Fluid and Cysts. 1. *Fluid.* This was faintly alkaline in reaction, of a straw-yellow color, and was clear except for a distinct opalescence when it was agitated, giving the impression that it contained a suspension of gold-dust. Chemically it contained a fair amount of albumin (coagulation by heat, potassium ferrocyanide, biuret tests, etc.), and a faint trace of grape sugar (Fehling's test). Sodium chloride was not present in excess. Tests for succinic acid and bile pigments were negative. Microscopically the fluid showed (*a*) *cholesterin* crystals in very large number, (*b*) occasional red blood-corpuscles (from the puncture, undoubtedly), (*c*) a few degenerated fatty cells, without nuclei, and (*d*) a few round, colorless bodies (4–18 μ), resembling crystals, that did not dissolve or effervesce on the addition of HCl. No echinococcus scolices, hooklets, calcareous corpuscles, or laminated cyst-membrane could be found in a search continued many days. The three specimens of fluid aspirated at different times were all similar in every respect. The specific gravity of the second specimen was 1026.

2. *Cysts.* The mass removed from the pleural cavity at operation consisted of a small mass of collapsed cysts closely adherent to one another, and varying in size from that of a millet-seed to that of a small lemon. The different cysts were, in general, easily separated by gentle traction, though some of them were so closely united that they could not be separated without rupturing their walls. Many of the cysts were found to spring from the walls of adjoining cysts, having a wall in common at their line of union. In the largest cyst, extending inward from its outer wall, were seen two or three small cysts, giving the appearance of daughter-cysts, but no free daughter-cysts were found. The number of individual cysts was not counted, but appeared to be approximately forty. Externally the cyst-walls looked very thin and delicate, white, translucent, and glistened with the impregnation of crystals. When cut the cyst-wall showed no tendency to curl. The interior of the cysts showed a relatively thick, soft, white, granular layer, which could be removed by the slightest touch. The cysts were all collapsed and without fluid content. When placed in water the cysts absorbed it and swelled to a globular form.

The *microscopical examination* of the fresh cysts showed as follows: (*a*) The *external wall*, when teased, showed no true lamination, though

slight suggestions of it were seen in occasional bands of fibrils. No cell structure could be made out. The tissue looked hyaline and structureless. Abundant cholesterin crystals were seen throughout. Treatment with dilute acetic acid and potassium hydrate solution did not modify essentially the appearance. Frozen sections stained with hæmatoxylin and eosin showed the same hyaline appearance, with many bands or striations branching off toward the interior of the cyst. On the surface an occasional degenerated cell nucleus was seen, but no true organized cellular membrane. (*b*) The *soft material lining the cysts* was found to consist chiefly of fatty detritus aud cholesterin crystals. There were many degenerated fatty cells, without nuclei, which stained well with Sudan III. An occasional, small, round, colorless, crystalline body was also seen, which did not dissolve or effervesce in dilute HCl. An exhaustive search, lasting for many days, for echinococcus scolices, hooklets, or calcareous corpuscles, was without reward for our labor, but Dr. Stiles, to whom specimens were submitted (see below), was more fortunate in his examination, and succeded in finding in the cyst content two *echinococcus hooklets.*

Sections of the cysts hardened in alcohol and formalin offered no evidence not obtained from the frozen sections. The histological examination of the walls of the small daughter-cysts protruding toward the interior of the largest cyst showed exactly the same structure as found in the larger external cysts, except that the few cells and nuclear fragments found at the periphery of the outer walls of the external cysts were not found over the walls of the daughter-cysts.

Believing, as we did, after a careful study of the case, that we were dealing with an atypical and rare form of degenerated echinococcus cysts, and before we had obtained conclusive support of our diagnosis, we submitted specimens of the fresh cysts and also stained sections of them to Professor William H. Welch, of Baltimore, and to Dr. Charles Wardell Stiles, Zoölogist of the Bureau of Animal Industry, United States Department of Agriculture, Washington, D. C., for their expert opinion. Extracts from their reports are here given, with their kind permission, as follows:

REPORTS OF PROF. WILLIAM H. WELCH.

"I have examined the sections of the cysts. I am of the opinion that the cysts are degenerated echinococcus cysts. The gross appearances of the specimens which you showed me were like those of echinococcus cysts. The microscopical characters of the fresh teased material and of sections of the hardened specimens seem to be best explained upon the assumption that the cysts are much degenerated hydatids. Cholesterin crystals and fatty detritus such as were present in these cysts are common in degenerated hydatids. No organized tissue is present in the walls of the cysts. These walls, as shown in the sections, present an outer thin hyaline membrane, with irregular inner surface continuous with fragments and shreds of a structureless material, which occupies a considerable part of the interior of the cysts on the sections. There are scarcely any intact cells in the sections; here and there a few are attached to the outer wall and a few nuclear fragments are seen in the interior, but there is nothing indicative of suppuration or of previously organized tissue, nor is there any suggestion of fibrin or inflammatory exudate in the sections.

"As no hooklets can be detected, I have looked carefully for the characteristic lamellation of the cuticular layer of echinococcus cysts. I think that there are *suggestions* of such lamellation in the parallel striæ occasionally seen in some of the more coherent and hyaline membranous fragments attached to the wall and in the interior of the cysts, but they are not so distinct as to be convincing.

"It is well known that echinococcus cysts may perish and undergo complete disintegration. Davaine, Leuckart, Neisser and others have described such degenerative changes in the dead cysts. In some of these cases nothing remains but the hooklets to establish the diagnosis. Sterile echinococcus cysts occur, and should these undergo similar degeneration it is evident that not even hooklets would be present to aid in the diagnosis.

"In your case the degeneration has not reached a stage in which the gross appearances of echinococcus cysts have been obliterated, but the characteristic physical and microscopical features do not seem to me reconcilable with any other sort of cysts than much degenerated echinococcus cysts. As already mentioned, the large amount of fatty detritus and of cholesterin crystals within the cysts is in favor of the diagnosis of degenerated hydatids.

"Echinococcus cysts, of course, receive their nourishment from the tissues in which they are embedded, and impairment of this nutrition tends to the production of sterility and degeneration of the cysts. In your case the partial and small attachment of the cysts to the pleural membrane must have favored these results of imperfect nutrition, and in this way I should explain, at least in part, the sterility of the cysts and their degenerated condition." (April 1, 1900.)

"In my previous letter I did not speak particularly of the outward curling of incised echinococcus cysts. This physical property is well known and is demonstrable readily in fresh, undegenerated cysts. As it depends upon the integrity of the parasite, it, of course, is likely to disappear after the parasite dies and undergoes degeneration, as certainly occurred in your case, assuming it to be a bladder-worm. It would not be contended, of course, that the absence of this property would exclude the diagnosis of a degenerated echinococcus cyst.

"As I wrote you, I attach more importance in explaining the sterility of the cysts and the evidence of death and degeneration of the parasite to the comparatively limited attachment of the cysts to surrounding living structures and their consequent feeble nutrition. Most of the instances of pleural echinococci have been of single or few large cysts, firmly attached in their circumference. The topographical relations of the cysts in your case were rather peculiar and unusual." (April 5, 1900.)

REPORT OF DR. CHARLES WARDELL STILES.

"Referring again to your letters of April 5th and 15th, I have this morning made a microscopical examination of the material you sent me, and all doubts have now been removed from my mind regarding the case, for in one preparation I succeeded in finding an *echinococcus* hook. In endeavoring to isolate it and mount it for you I unfortunately lost it, and could not again find it.

"As for the interpretation of the cysts which you showed me, you will recall that at the time I examined them they did not make the

impression upon me of being hydatid cysts, nor did your microscopical preparations convince me that they were such. They certainly did not look like any echinococcus cysts which I have ever seen before, nor did they behave as such. This, however, was purely a negative opinion, and the evidence both for and against the interpretation appeared to me to be so uncertain that I was inclined to take entirely a neutral position in this regard. I have examined hundreds of specimens of this parasite from various animals, and I believe this is the first time that I have ever felt in doubt regarding the cyst itself. Finding the hook, however, removes all doubt in my mind relative to the existence at one time of a hydatid cyst in the structure." (April 18, 1900.)

It is seen from the very critical and full reports of Professor Welch and Dr. Stiles, as well as from the report of our own study of the tissues, above given, that this case was one of unusual pathological interest and rarity, and for this reason we have considered it worthy of a rather full and detailed narration of its study, step by step, through all the difficulties attached to its correct interpretation to its final and definite diagnosis. That the case was one of degeneration of hydatid cysts we entertained hardly a doubt after considering all the available evidence, but the proof of this, convincing to others, was not fully in hand until Dr. Stiles' fortunate discovery of a single[1] echinococcus hooklet.

As to the interpretation of the nature and origin of the outer cyst wall, *i. e.*, whether this thin, hyaline, structureless membrane was derived by a process of degeneration from the cuticular wall of the parasite itself or from the once cellular, fibrous capsule furnished by the surrounding tissues of the host, all doubt seems to be definitely removed by the study of the walls of the small cysts (daughter-cysts) found protruding into the cavity of the largest external cyst. Sections made through that portion of the daughter-cysts which was directed toward the interior of the large cyst showed its wall to be constituted of exactly the same hyaline, structureless membrane which formed the substance of the outer wall of the larger and separate external cysts. The lining membrane, also, of the internal daughter-cysts was identical in its histological structure with that of the external cysts. The only point of difference found in the finer anatomy between the daughter-cysts and the external cysts was the fact that the former failed to show the occasional degenerated cells and nuclear fragments found at the periphery of the latter. These facts make it clear, we think, that the hyaline, structureless membrane of the outer cyst-walls was derived by a process of degeneration from the cuticle of the parasite itself and not from the tissues of the host.

As Professor Welch has clearly stated, what little is known of the general causes leading to the death and degeneration of the bladder-worm, we need not dwell upon this feature of the case further than to

[1] Dr. Stiles writes that he has since succeeded in finding a second booklet.

state more explicitly than we have previously done, the fact that the mass of cysts removed at operation seemed to be attached to the costal pleura in the most delicate manner, by which its removal was effected without appreciable resistance and almost by chance. This loose or partial attachment of the cysts and the churning motion to which they were subjected by the respiratory movements of the lung doubtless determined largely their degeneration.

In reviewing the various cases of echinococcus of the pleura reported in the literature, we have found no mention of a type of degeneration of the cyst-walls similar to that found in our case, nor have we been able to find any reference to such a process of degeneration in the walls of echinococcus cysts from other parts of the body. It is a fact, however, that the reports of cases only occasionally describe the histological structure of the cyst-walls, so that it is possible that the loss of the characteristic lamellation of the outer wall and its degeneration into a thin, structureless, hyaline membrane, *without gross degeneration*, may not be wholly unknown. Such a process, however, must be exceedingly uncommon, else it would have been described in the standard works on helminthology. Dr. Stiles, whose reputation entitles him to recognition as the leading helminthologist in America, states that he has never before, among hundreds of cases examined, encountered a case in which he was in doubt regarding the cyst itself.

We, therefore, assume that in reporting this case, we may, perhaps, be placing on record for the first time the type of degeneration of echinococcus walls which we have above described. Various other processes of degeneration of echinococci have been described, of which may be stated the following brief summary: The earliest changes usually begin in the surrounding capsule, which softens and disintegrates, or, on the other hand, hypertrophies or undergoes calcareous degeneration. This is followed by the fatty degeneration and disintegration of the parenchyma lining the interior of the cyst. The fluid contents of the cyst are gradually absorbed and inspissated into a granular and fatty detritus, or may undergo gelatiniform, colloid, caseous, or purulent degeneration, while the cuticle softens and disintegrates into an amorphous detritus, or may become calcified. These processes may also follow the rupture of the cyst. Sometimes the process of degeneration is so complete that there is left only a small, dried-up, knotty mass as the remains of the previous cyst.

The study of the fluid aspirated from the cysts did not assist materially in reaching a diagnosis. Hooklets, scolices, calcareous corpuscles and bits of cyst-membrane were all absent, and thus spoke against echinococcus fluid. It must be remembered, however, that these structures are found only occasionally in the cyst fluid, according to Leuckart,[1]

[1] Die Parasiten des Menschen., 2 Aufl., Bd. i., 1886, Leipzig and Heidelberg.

"only in a very small minority of the cases of human echinococcus." Hooklets were mentioned in only seven out of the sixty-seven cases of hydatid disease collected by Sommer[1] from the United States. The high specific gravity (1026) of the fluid and its considerable amount of albumin were opposed to the diagnosis of echinococcus. The specific gravity of hydatid fluid ranges usually from 1005 to 1013, and albumin is said by most observers to be absent. Still, instances of high specific gravity and abundant albumin in hydatid fluid are recorded, especially in cases of degenerated hydatids. Cordier,[2] in a case of sterile echinococcus cyst of the liver, found a "serosanguinolent" fluid containing albumin and having a specific gravity of 1032. Bruce and Sheild,[3] in a case of gelatiniform degeneration of hydatid cysts of the liver, found a gelatinous fluid that was "highly albuminous." Mosler and Peiper[4] have found albumin in such cases of hydatids only as were influenced by a surrounding inflammatory process. Neisser[5] states that after repeated exploratory punctures the fluid may contain albumin. Knaggs[6] found a considerable amount of albumin in the fluid withdrawn from a hydatid cyst by the first puncture. Rosenstein[7] and Jaeger[8] have claimed even that traces of albumin were the rule in hydatid fluid. The presence of succinic acid is regarded as pathognomonic of hydatid fluid, but its absence is of no value as evidence. This acid is said by Leuckart to be "found in hardly any other living organism." Traces of grape sugar, as in our case, are usually found. The presence of cholesterin crystals, in large amount, is certainly suggestive of degenerated hydatids. Still, caution must be enjoined in using this point for diagnosis, for a considerable number of cholesterin crystals may be encountered in fluid from various other cysts and encysted conditions in the body, as we have recently seen in a case of degenerated simple cyst of the testicle and in a case of colloid carcinoma of the peritoneum. The large amount of cholesterin crystals in the fluid did, however, and correctly, lead us to the opinion that the fluid in the chest was at least encysted, though, as we erroneously supposed, only by inflammatory adhesions and fibrous tissue. Thus, as is seen, the study of the fluid offered little evidence for the diagnosis of hydatid cyst.

We have assumed that the fluid obtained by puncture of the chest cavity came, in fact, from the cysts. This conclusion could hardly be controverted, we think, after considering the facts above set forth. In this connection it may be worth while to explain the difficulty that

[1] Echinococcus Diseases in the United States. Medical Record, New York, Nov. 23, 1895.
[2] Langsdale's Lancet, Kansas City, April, 1896.
[3] Med.-Chir. Trans., London, 1891-1892, vol. lxxv. pp. 175-188.
[4] Thierische Parasiten. Wien, 1894. [5] Die Echinococcen-Krankheit. Berlin, 1877.
[6] Schmidt's Jahrbücher, clxiv., p. 198.
[7] Die Pathol. und Therap. der Harnkrankheiten. Berlin, 1886.
[8] Meckel's Archiv, Bd. vi.

was encountered in obtaining fluid by puncture over the area of dulness. On the first occasion the needle had to be inserted five times before fluid was withdrawn, and on the second occasion also five separate punctures were made before fluid was obtained. The very loose attachment of the cysts to the costal pleura and the impregnation of their hyaline walls with crystals of cholesterin, forming a shield over them, as it were, appear to us to be sufficient to explain this fact.

As the physical examination, symptoms, and history of the case offered no evidence of the past or present existence of hydatids in other organs, we have assumed the cysts of the pleura to be primary. *Primary echinococcus cysts of the pleura* are of very rare occurrence and we have succeeded in finding only thirty-nine cases in the literature.[1] These thirty-nine cases include Neisser's[2] seventeen cases, Madelung's[3] one case, Rosenthal's[4] review of five cases, Maydl's[5] collection of four additional cases, Winzerling's[6] one case and his two additional cases found in the literature, Mosler and Peiper's[7] one case, Ascoli's[8] four cases, Vannini's[9] one case, Pasca's[10] one case, Orlandi's[11] one case, and von Bókay's[12] one case. Adding our own case to this enumeration, we have, then, *a total of forty recorded cases of primary echinococcus of the pleura.* Of these forty cases, thirty-two were pleural and eight were peripleural; twenty-four occurred on the right side, eight on the left side, and in eight cases the side was not stated. Echinococci of the pleura secondary to the disease in neighboring organs, especially the liver, are of more frequent occurrence, though still very uncommon.

The exogenous form of echinococcus cysts (*Exogener echinococcus*, Kuhn), as seen in our case, is relatively uncommon in man, at least in its typical form of multiple, external budding. The endogenous form (*Echinococcus hydatidosus*, Leuckart) and the simple, single cysts are the forms usually seen in man. In herbivorous animals the exogenous form is said to be common (Leuckart). A very rare form of the exogenous hyatids is the *Echinococcus racemosus* (Leuckart), which differs from the commoner form of exogenous cysts only in the greater number and

[1] This collection of cases does not include the case reported by Dr. D. F. Smith, of Walkerton, Ontario, under the title "A Case of Hydatids of the Lung" (Canada Medical and Surgical Journal, November, 1882, xi. pp. 195–197). The description of this case leads us to believe that it was one probably of primary cyst of the pleura, of the peripleural variety.

[2] Loc. cit.

[3] Beitrage Mecklenbg. Aerzte zur Lehre der Echinokokkenkrankheit. Stuttgart, 1885.

[4] Ueber den Echinococcus der Muskeln. Diss., Berlin, 1888.

[5] Ueber Echinokokkus der Pleura. Wien., 1891.

[6] Ein Beitrag zur Casuistik des primären Pleuraechinococcus. Dissert., Jena, 1892.

[7] Loc. cit.

[8] La Riforma medica, Napoli, 1894, p. 729.

[9] Bullettino delle Scienze Mediche, Bologna, May, 1896, p. 240.

[10] Bull. del. Soc. Lancis. deg. Osped. di Roma. 1896, anno xvi., fasc. ii. p. 84.

[11] Gazz. Med. di Torino, 1898, vol. xlix. pp. 501, 521, and 565.

[12] Jacobi's Festschrift. New York, 1900, p. 211.

smaller size of the cysts, which resemble a bunch of tiny grapes. Indeed, were the individual cysts of our case all small, we might properly describe them as racemose hydatids. No case of multiple, budding, exogenous echinococcus cysts of the pleura has been previously described, so far as we have been able to ascertain.

The physical signs found at the base of the right chest in our case were not especially suggestive of echinococcus cysts as opposed to encysted pleural effusion, though no positive, differential, physical signs are known, save the "*fremissement hydatique*" first described by Briançon[1] and Piorry[2], which could hardly be expected to be obtained in its typical form through the bony chest-wall. It is possible, however, that the prolonged, harsh, vibrating fremitus which we felt and heard over the area of dulness was in fact a *modified hydatid fremitus* produced by the movements imparted to the cysts by the lung in its inspiratory expansion. An irregular line of limitation of the percussion flatness has been said to be usual in hydatid cysts of the pleura, as well as an anomalous mingling of normal and abnormal physical signs on palpation and auscultation. As illustrated by our case, there seems to be little tendency for echinococci of the pleura to invade by extension neighboring organs, excepting in the case of peripleural echinococci, and curiously, also, echinococci of the pleura rarely excite pleurisy.

The prognosis in unoperated hydatid cysts of the pleura, primary and secondary, is almost hopeless, and no remedial measure short of radical extirpatory operation should be considered. Of the thirty-one unoperated cases of primary and secondary echinococci of the pleura in Neisser's statistics, all died without exception, including even those cases in which evacuation took place by rupture into a bronchus. The fatality of unoperated hydatid disease of the pleura is accounted for in part by the tendency of the cysts to become infected and purulent, and also, in many cases, by the profound *toxæmia* resulting from the rupture of the cysts and the absorption of their fluid, which is very toxic. Brieger[3] first and later, also, Boinet and Chazovlière[4] have succeeded in separating from hydatid fluid a toxic substance that is rapidly fatal to animals after injection. For the same reason exploratory puncture of the cysts has been followed in many cases by a rapidly developing toxæmia and death, as well as by sepsis. A common sign of toxæmia following exploratory puncture is the development of a general urticarial eruption. Because of the danger of sepsis and toxæmia resulting, it would be conservative practice to undertake a radical operation, following ex-

[1] Essai sur le diagnostic et le traitment des acéphalocystes. Thesis, Paris, August 26, 1828.
[2] De la percussion médiate, et des signes obtenus à l'aide de ce nouveau moyen d'exploration. Paris, 1828.
[3] Langenbuch's Leber-echinococcus und seine Chirurgie, p. 78.
[4] Revue de Medécine, November, 1898, No. 11.

ploratory puncture, at the very earliest possible moment after reaching a diagnosis of echinococcus cyst.

In conclusion, we wish to express to Professor William H. Welch and Dr. Charles Wardell Stiles our great indebtedness for their interest in our case and their valuable assistance in confirming our diagnosis.

SARCOMA AND CIRRHOSIS OF THE LIVER.

By W. W. Ford, M.D., D.P.H.,
FELLOW IN PATHOLOGY, M'GILL UNIVERSITY, MONTREAL.

(From the Pathological Laboratory of the Montreal General Hospital.)

The combination of carcinoma and cirrhosis of the liver was pointed out by Finley and Adami in the *Montreal Medical Journal*, April, 1895, when they reported a case which showed these two different pathological conditions. Their case was followed by a paper by Fussell and Kelly, of Philadelphia, read before the Association of American Physicians, in which two other cases of a similar combination were presented.

The association of cirrhosis of the liver and carcinoma of this organ is readily explained when we remember the presence in the liver of many structures epithelial in character. The association of sarcoma and cirrhosis of the liver is possibly rarer than the association of carcinoma and cirrhosis, and the presence of the former variety of tumor is more difficult to explain than the presence of the latter.

The following case, which occurred in Dr. Finley's wards in the Montreal General Hospital, and which by his kind permission is here reported, represents a combination of an extreme grade of cirrhosis of the liver, with a large tumor mass in the right lobe, made up of sarcomatous tissues, and a number of secondary nodules scattered throughout the peritoneal cavity.

History. L. P., male, aged fifty-nine years, admitted February 15, 1900. When admitted the patient was incoherent, with a thick, halting speech, and quite unable to give any clear account of his illness. The meagre facts gained on careful questioning showed that he had used alcohol, in the form of gin and whiskey, to excess for a number of years, and had suffered from comparatively no illness of any duration up to his present attack. He stated that for three or four months he had a slight cough, with a little expectoration. He left work a month ago, because, he said, of cold sensations.

Three or four days before admission the patient's sister died, and he went on a long spree, lasting for forty-eight hours. The day before entrance into the hospital, while the patient was being shaved, he had an attack of cerebral paralysis, after which he was unable to raise his left arm or to move his left leg. He suffered from left-sided headache at the

same time. A swelling of the abdomen, which the patient said began several days before the paralysis, increased with great rapidity subsequent to this seizure.

Condition on Admission. Patient is a man aged about sixty-five years, whose speech is so thick and indistinct as to render answers to questions almost unintelligible. He is extremely drowsy, the skin is muddy, and there are a few stellate veins about the nose, cheeks and forehead; subcutaneous fat moderate in extent, and muscles of fair size and firmness. The abdomen is greatly distended; the surface is covered with scratch marks, showing a slight dilatation of the superficial veins. There is dulness in both flanks, which changes to resonance with the shifting of the patient's body. The liver dulness extends from the fifth rib to the costal border, measuring three inches in nipple line; neither liver nor spleen is palpable.

The heart sounds are distant and feeble, apex impulse not palpable; pulse small and feeble, 120 to the minute. There is slight cyanosis of finger nails.

Complete left hemiplegia, the face being slightly drawn to the right side, the left labio-nasal fold shallower than the right. The left hand and arm and the left leg are quite powerless, the arm is held flexed across the chest; there is no rigidity of either extremity. The patellar reflexes are increased, the plantar present on the right side, absent on the left.

The day after admission the patient's condition became much worse. He developed great restlessness, and a dull, drowsy stupor; articulation was greatly impaired, and the pulse much diminished in strength and frequency; temperature between 97° and 98°. The patient died about midnight of the second day.

Diagnosis during Life. Cirrhosis of the liver and abdominal ascites, left-sided hemiplegia, and right-sided thrombosis of the cerebral vessels.

The autopsy was performed the day following the death of the patient.

Autopsy Notes. Body of a large, well-developed man of medium height; no jaundice; lips and mucous membranes pale; mouth and pharynx filled with clear watery fluid; marked lividity of dependent parts of the body, and complete rigor mortis in arms and legs; no petechia of skin; external genitalia normal.

Thorax: A few pleural adhesions at apex of right lung. Hypostatic congestion of both bases with slight acute inflammation of the walls of the larger bronchi. No areas of pneumonia or tuberculous consolidation. Pleural cavities free from fluid.

Larynx: Congested and slightly œdematous.

Thyroid: Normal.

Heart: Not enlarged; no pericardial adhesions; a thick layer of pericardial fat. Mitral valves thickened; aortic valves normal. Aorta not calcified. Heart muscle brownish.

Abdomen contains 4000 c.c., pale-yellow ascitic fluid, not blood-stained. Mesentery and omentum filled with small nodules the size of a pea, white or reddish; similar nodules scattered over peritoneal surface of abdominal muscles and of the diaphragm. The omentum was so filled with these nodules as to form a hard, stiff, ridge-like swelling, palpable just below the costal margin. Stomach and intestines normal; rectum normal.

Liver: The edge does not reach the costal margin. It is considerably shrunken in size, the surface hard, very rough, and granular. About the middle of the liver is a band of more nearly normal hepatic parenchyma which projects some distance from the otherwise hard and cirrhotic organ. On section the liver cuts with great difficulty, the fibrous tissue being greatly increased. The fibrosis is irregular in distribution, practically *no* normal hepatic tissue being left. The cut surface is a pale gray in color. In the right lobe of the liver is a large tumor mass about 5x8 cm. in dimensions. About a soft, white friable centre is a dense, hard peripheral zone, grayish-white in color.

Portal vein normal; no thrombosis.

Kidneys: Small, contracted, capsule strips off with great difficulty, leaving a slightly roughened, puckered surface. On section the fibrous tissue is much increased.

Pancreas: Normal; no hemorrhages.

Spleen: Not enlarged; capsule is soft, pale, and smooth. On section cortex and medulla are paler than normal.

Suprarenals: Normal.

Bladder: Contracted and muscle-wall thickened; mucous membrane normal.

Prostate: Enlarged and fibrous.

Brain: Meninges normal. The Sylvian artery on the left side just at its branching is occupied by a rather firm thrombus, whitish or grayish-white in color, slightly adherent to the walls of the vessel. On section the brain is normal. No areas of softening to be made out in any portions.

Microscopical Examination of the Liver. Microscopical examination of various portions of the liver reveals almost pure fibrous tissue infiltrated with small round cells and small spindle cells, only a few liver cells being left intact. In some areas there is a well-marked fatty degeneration of the liver cells, with considerable injection of the capillaries where the liver lobules can can be made out; the cirrhosis is seen to be *interlobular* in character, while in much of the organ the lobules have been quite destroyed by the new tissue, thus representing a condition of *intralobular* cirrhosis as well.

Microscopical examination of the tumor in the right lobe of the liver shows the central part to be composed of necrosed, broken-down, sarcomatous bodies, and the periphery to consist of dense fibrous tissue infiltrated with small round and spindle cells. The central part stains badly, but shows chiefly sarcomatous cells—both spindle cells and round cells.

Nodules in the peritoneum are found to be composed of similar spindle cells, with many bloodvessels scattered through the matrix of fibrous tissue.

Anatomical Diagnosis. Hypostatic congestion of lungs; congestion of larynx and trachea; sclerosis of mitral valves; cirrhosis and sarcoma of liver, with secondary nodules in mesentery, omentum, and peritoneum; hypertrophy of prostate and of muscle-wall of the bladder; thrombosis of Sylvian artery, left side.

The presence of cirrhosis of the liver in this case is explained by the intemperate habits of the patient, who had evidently been a chronic alcoholic. The liver corresponds in type to the ordinary *atrophic cir-*

rhosis of Lænnec or the alcoholic cirrhosis, and the clinical history of the patient and the development of ascites are both explained by the condition of this organ.

The presence of the huge mass of sarcomatous tissue in the right lobe of the liver is somewhat more difficult to explain. It is a definite sarcoma of a combination of round and spindle cells. Sarcoma of the liver occurs in two varieties, primary and secondary. The primary sarcomas are exceedingly rare, and it has often been doubted by pathologists whether they ever do occur. Genuine cases are recorded, however, by Horup, Lancereaux, Arnold, Windrath, Waring, von Kahlden and others. Such tumors are said to arise from the walls of the portal vein and from the smaller bloodvessels, and von Kahlden states that frequently older cirrhotic changes in the liver exist as well. While the presence of cirrhotic changes is admitted by Quincke and Hoppe-Seyler to be a possible factor in causing the development of *carcinoma* of the liver, they do not admit that a similar cirrhotic condition is capable of producing *sarcomatous* growths. Those writers who believe that cirrhosis may cause sarcoma, do not quote any cases in which such a definite combination has been seen.

Secondary sarcomas occur in the form of metastatic tumor nodules scattered through the substance of the liver, or in the form of a diffuse sarcomatous infiltration. The primary seat of such a tumor is usually the skin or the choriod coat of the eye. The most frequent variety of secondary sarcomas is the melanotic sarcoma, the metastases of which give the liver a dark-brown or black appearance on section. The present sarcoma was not melanotic, and the pigmented coat of the eye and of the skin may be eliminated as a primary seat for this growth.

Its presence as a large tumor nodule in one lobe of the liver, unassociated with any other nodules of this organ and unassociated with any tumor nodules in other parts of the body, except the small metastases in the peritoneum and omentum, point to its being possibly primary in the liver. This cannot be stated with positiveness, however, for it is impossible to eliminate as the primary cause of any tumor masses in the liver a tumor of some one of the other organs, or some variety of cutaneous tumor which might have been removed years prior to the autopsy. It is a common occurrence in large autopsy-rooms to find secondary tumor masses in the internal organs of the body which give comparatively few symptoms during life, and which are not necessarily the cause of death, the presence of which is explained only by the discovery that the patient had, possibly some years previous to his death, some tumor removed from the skin or subcutaneous tissues.

The pea-sized bodies in the omentum and in the peritoneum were evidently derived from the tumor of the liver, or possibly from that tumor, which might have been the primary seat of both. At any rate,

the presence of the sarcomatous mass in the liver is not explained unless it be considered as a primary tumor, and its association with cirrhosis of this organ must be considered quite unique. The examination of the literature of cirrhosis of the liver, especially the standard textbooks and the various medical indices, fails to reveal any quotations of a similar combination of sarcoma and cirrhosis, although such cases have doubtless been already reported, possibly under different names, yet the combination represented by the present case is, as far as we know, the first case to be reported in this country.

REPORT OF A CASE OF MELÆNA NEONATORUM DUE APPARENTLY TO AN INFECTION BY THE BACILLUS PYOCYANEUS.

By William R. Nicholson, Jr., Ph.B., M.D.,
ATTENDING OBSTETRICIAN TO THE MATERNITY HOSPITAL OF PHILADELPHIA; ASSISTANT DEMONSTRATOR OF OBSTETRICS AND INSTRUCTOR IN GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA; ASSISTANT SURGEON TO THE GYNECEAN HOSPITAL.

The case, the subject of this report, is that of a boy born at term in the Maternity Hospital of Philadelphia. Presentation was of the vertex, and the labor lasted but six hours. Weight of child at birth was 3350 grammes. The child seemed perfectly healthy, and the cord separated without incident, except that the period between birth and separation was decidedly shorter than usual. Nothing abnormal was noted in the condition of the child until the sixteenth day, when the resident physician called my attention to it because its mouth had become sore. On examination the condition of the mouth was seen to be due to a slight stomatitis. There was no temperature rise. The next day, while the mouth did not seem to have improved as much as I expected, and a little blood was noticeable upon the lips, the condition of the child was not apparently at all changed for the worse, except that there was a slight elevation of temperature and that its bowel movements had become green. On the following day, however, the whole appearance of the child had markedly changed. The temperature, while not excessively high, was distinctly febrile, and the slight trace of blood noticed previously had become a constant flow, apparently from the mucous membrane of the entire mouth. The bleeding was so profuse that all attempts to examine the mouth and pharynx were unavailing. The child, indeed, had to be watched carefully to prevent embarrassment of the breathing from the constantly flowing blood. Decided loss of strength was apparent. The lungs were negative. All attempts at nursing were futile. The stools at this time contained blood in considerable quantity and of a bright red color. The condition continued unchanged, except for the occurrence of a few convulsions, until the next day, the nineteenth since birth, when death took place.

The clinical diagnosis was simply melæna neonatorum, as during life there were no facts at command which would enable a more definite

diagnosis to be made. The possibility of its being a case of hæmophilia occurred to me, but the age of the child, the association of febrile temperature, and the absence of history, seemed to preclude any etiological relationship.

The post-mortem examination was made by the pathologist of the Maternity Hospital, Dr. Joseph Sailer, to whom I desire to express my most sincere thanks.

The notes were elaborate, and from them I have abstracted the most important facts.

The body was decidedly emaciated. A large, distinct, hemorrhagic effusion noticed around the umbilicus.

Abdominal cavity: No subcutaneous fat. Hemorrhage noted into the wall of the umbilical cord, and hemorrhagic effusion into the peritoneum. The cæcum was distended and somewhat adherent to the abdominal wall. In the neighborhood of the umbilical artery the peritoneum was dry and sticky. The ileum was of a slaty color, due to contained blood. The mesentery was intensely congested. The mesenteric glands not enlarged. No ecchymotic hemorrhages.

Thoracic cavity: Pleural cavities were free and contained no fluid. The pericardium was smooth, but intensely congested, particularly along the line of the anterior coronary artery.

The lungs: Pale. Numerous subpleural ecchymoses, and some areas of consolidation were found on the right side.

The heart: The right auricle was distended with blood. The foramen ovale was widely patulous. No coronary valve was present. The ductus arteriosus was not investigated. Otherwise the heart was normal.

The spleen: Normal.

The ileum: Filled with a dark-brown granular mass, which was readily removed, leaving a smooth, glistening and pale mucous membrane which showed no congestion. The folds of the ileocæcal valve contained a firm, slightly adherent clot. There was no sign of ulceration in the intestinal tract. The common bile-duct was patulous. The mucous membrane of the duodenum was clear and but slightly bile-stained.

The stomach contained a small amount of bile, but no blood. The mucous membrane was very rugous. There were no ecchymoses.

The œsophagus was filled with blood-stained mucous.

The liver was enlarged and soft, and its surface on section was distinctly cloudy. The structure was indistinct.

The pancreas was soft. There was no hemorrhage present, and its structure appeared to be normal.

The adrenals and kidneys were both apparently normal.

The brain was apparently œdematous. The membranes were very cloudy. There were no hemorrhages either into the substance or ventricles.

The histological and bacteriological examinations were made by Dr. Sailer in the Pathological Laboratory of the University of Pennsylvania.

On section through the duodenum some superficial necrosis in the mucous membrane was present, and also moderate proliferation of the epithelium cells of the villi. Very slight perivascular round-cell infiltration in the submucosa. The muscularis was normal. There was a slight exudate on serous coat. High magnification gave some cloudy swelling in the epithelial cells of the villi, and many of the cells were seen to contain swollen and pale nuclei. The walls of the bloodvessels were found to be slightly thickened.

The peritoneal exudate contained some rod-shaped micro-organisms in the superficial necrotic areas of the mucous membrane. In this area there were some cells whose nuclei were large and stained deeply, the protoplasm of the cells also taking a portion of the stain.

The pancreas showed a distinct hyperplasia of the connective tissue surrounding the bloodvessels, with some round-cell infiltration. The ducts were lined with apparently normal cylindrical epithelium, with deeply staining nuclei between the lobules. The connective tissue was generally hyperplastic, and showed here and there a few accumulations of round cells. The bloodvessels were distended with blood and their walls were distinctly thickened. In some of them the intima was detached and in others distinctly swollen. The media was slightly hypertrophied, but the thickening affected chiefly the adventitia, which was usually about twice the thickness of the media. The walls of the veins were distinctly thickened and surrounded by a mass of fibrous connective tissue. The cells of the pancreas stained very irregularly. In many of them there was distinct alteration of the protoplasm which stained deeply with eosin. In many places the granular acini were pushed apart by dense infiltration of cells having round or oval nuclei. The bodies of Langerhans were numerous, frequently slightly lobulated, and in some places there was apparently degeneration in the cells, the nuclei being enlarged, irregular, and staining very deeply, the protoplasm retaining the hæmatoxylin stain. Staining by thionin gave essentially the same changes. No micro-organisms could be detected in the tissues. Occasional eosinophile cells were observed in the midst of the cellular infiltrate. Many of the nuclei of the cells of the pancreas showed extreme vacuolation. In some cases two deeply staining nuclei were found in the same cell. Vacuoles also occurred in the cellular protoplasm.

The liver showed the same hyperplasia of the connective tissue, with moderate round-cell infiltration, particularly perivascular, as in the pancreas. The bloodvessels were distended. The bile ducts were apparently normal. Here and there the substance of the liver was infiltrated with red blood cells. The small vessels of the substance were surrounded by round cells possessing a single round vesicular nucleus. The arterial walls were greatly thickened, particularly the adventitia. The walls of the veins were also considerably dilated and surrounded by connective tissue. The nuclei of the liver cells were all about of equal size and stained distinctly with a clear chromatin. In addition, there were a number of small cells with deeply staining nuclei, and some larger round cells, the protoplasm of which retained the hæmatoxylin stain. With immersion the nuclei could be seen to have the normal structure. The small dark cells were evidently polynuclear leucocytes. There were in addition certain cells showing fragmentation of the nuclei. The protoplasm of the liver cells took eosin stain faintly. There were some clear globules in the tissue. The radicles of the hepatic vein were distinctly distended, but were apparently normal. The portal vessels were also distended, but not markedly. The protoplasm of many of the hepatic cells appeared to be slightly granular. There were no organisms found in the tissues. The intestines showed a moderate necrosis of the superficial layer of the mucosa and some thickening of the submucosa, with considerable round-cell infiltration about the glands. The serous coat appeared to be normal. The epithelial cells were in part desquamated, and appeared to have proliferated. The epithelial cells of the crypts

were normal. The bloodvessels showed a slight thickening of the walls. The mucuous membrane contained a large quantity of rod-shaped bacilli.

The spleen: Trabeculæ were prominent, and the walls of the bloodvessels were tremendously thickened, particularly the adventitia. The perivascular connective tissue was also distinctly hyperplastic. In some areas there was intense perivascular round-cell infiltration. Throughout the greater part of the tissue in this section there was hemorrhagic diffusion, but other parts were relatively free. The nuclei of the cells stained distinctly, There was no evidence of giant-cell formation or of fragmentation. The capsule was about normal in thickness, but it sent several trabeculæ into the substance which were quite thick, and which branched more or less extensively. The presence of a very few rod-shaped organisms was determined in the substance.

The thymus showed slight degenerative changes, as did also the tail of the pancreas.

To recapitulate: There was found post-mortem a generalized parenchymatous degeneration of the organs, a sclerosis of the pancreas, an acute enteritis, and a commencing cirrhosis of the liver.

The perivasculitis, the cirrhosis of the liver, together with the sclerosis of the pancreas, approached the changes found in senility. Finally, it is to be especially noted that there were no other evidences of syphilis.

The presence of a chronic pancreatitis in a new-born child is, I believe, in the absence of syphilitic manifestations, of sufficient rarity to deserve at least passing notice. The usual cause of connective tissue overgrowth in this organ is, of course, syphilis, and when it was found I was somewhat inclined to believe that we might have been dealing with a case of that disease, in spite of the fact that there were no other signs.

In looking up the subject more carefully, however, I have found that the presence of a connective tissue hyperplasia in the pancreas has been noted in the new-born in various conditions. Kasahara,[1] in the Pathological Institute of the University of Berlin, made some particularly interesting studies of the condition. His article gives a list of cases in which this condition was found at post-mortem, and among his cases he was able to tabulate twenty-two in children whose ages varied from a few months to several years.

Among these children he found six in whom there was a marked hyperplasia of the pancreatic connective tissue, and four in whom it was moderately increased. The remaining twelve cases showed but the normal amount. In all of these cases the author says that the diseases causing death could in no case have been the cause of the interstitial pancreatitis, and he also excluded syphilis by most careful search for other signs. He is somewhat disposed to accept the condition as a

[1] Ueber das Bindegewebe des Pankreas bei verschiedenen Krankheiten. M. Kasahara. Kioto, Japan.

physiological one, and calls attention to the relative richness in connective tissue at times found in the pancreas in childhood.

The diseases which caused the death of these children were very dissimilar, as the subjoined tabulation shows:

CASE I. Child, aged eight months; cause of death, diphtheritic colitis.

CASE II. Child, aged two months; cause of death, catarrhal gastro-enteritis, hemorrhagic parenchymatous nephritis, with thrombosis of the left renal vein.

CASE III. Child, three months; cause of death, bronchopneumonia and atelectasis.

CASE IV. Child, aged two and one-half months; cause of death, catarrhal enteritis and pulmonary œdema.

CASE V. Child, aged one and one-half year; cause of death, marasmus.

CASE VI. Child, aged six months; cause of death, catarrhal enteritis, bronchitis, bronchopneumonia, parenchymatous nephritis.

CASE VII. Child, aged one and one-half year; cause of death, catarrhal gastro-enteritis and atelectasis.

CASE VIII. Child, aged eight years; cause of death, phthisis pulmonalis.

Bacteriological Examination. At autopsy cultures were made from the heart's blood, left lung, spleen, liver, peritoneum, brain, contents of the ileum, and the bile.

The staphylococcus pyogenes aureus was recognized by the color of its colonies and its staining and culture peculiarities in the heart's blood, spleen, and brain.

The bacillus ærogenes lactis was similarly determined in the heart's blood, spleen, and peritoneum. The latter was also found in plates made from the contents of the ileum.

From the bile two micro-organisms were obtained, the one a long bacillus forming gas with glucose, and turning litmus milk-white without coagulation. This bacillus did not liquefy gelatin, did not stain by Gram's method, and was non-motile. In all its peculiarities it resembled the bacillus ærogenes lactis. The other bacillus found in the bile was a typical growth of bacillus pyocyaneus. Its identity was determined by the characteristic waving motion, by the deep bluish-green staining of the media, by the dark-blue color which it produced in strongly alkaline media, and by the rapidity of its liquefaction of gelatin.

From the liver tissue there were obtained the bacillus ærogenes lactis, the staphylococcus aureus, and the bacillus pyocyaneus.

In the left lung only the staphylococcus aureus was found.

A guinea-pig was inoculated with fresh bouillon culture of the staphylococcus aureus. It died the next day, and growths were obtained from all its tissues. The lesions were characteristic. Another pig was inoculated with the bacillus ærogenes lactis, and died within twenty-four hours. Pure cultures of the micro-organism were obtained from its tissues.

A third pig subsequently inoculated with the bacillus pyocyaneus died within twenty-four hours. Cultures were obtained from all its tissues.

It appears, therefore, that these three micro-organisms, which were dis-

tributed more or less generally through the tissues, were without exception virulent.

To recapitulate: There were three acute infections, two of which, the bacillus ærogenes lactis and the staphylococcus pyogenes aureus, were general, while the third infection, due to the bacillus pyocyaneus, was local, it being found only in the tissues of the liver and in the bile. That all three were virulent was proven experimentally.

The comparatively recent time of making the post-mortem, together with the marked localization, would seem to do away with any question of post-mortem invasion of the bacillus pyocyaneus. Also, the presence of the bacillus ærogenes lactis in the heart's blood would prove that its invasion was ante-mortem, as otherwise, though in a normal inhabitant of the intestine, it would not have been found in the heart in the absence of advanced putrefaction, since it is a non-motile bacillus.

The bacillus pyocyaneus has been known since 1882, when Gessard, "La Pyocyanine," *Thèse de Paris*, for the first time proved that the blue or green stain at times noticed on surgical dressings, and which, as early as 1860, had been isolated in the form of a coloring principle by Fordos (*Compt. rend. Acad. des Sciences*, 1860), was due to this bacillus, which he for the first time isolated in pure culture. For a long time subsequently, however, the organism was considered as a saprophyte, and even after it was shown as the probable cause of local or external pathological conditions it was still held, as, for instance, by Schimmelbusch, that it was limited in its action to the exterior of the body.

Many observers have noted its presence in abscesses of the ear and mastoid disease, and it also has been found in the pus of wounds upon the surface of the body. Its occurrence, however, upon the surface of the body cannot be considered common, as Jakowski was only able to find it twice in 200 cases.[1] It has also been found in tubercular cavities by Koch. Babes[2] reported a localized infection of the umbilical cord as due to it. Other investigators have recently found it within the body, as, for example, Barker (*Journal of the American Medical Association*, 1897), who determined its presence in an ovarian abscess, and Lartigau,[3] in purulent peritonitis. Charrin, 1894, reported its presence associated in puerperal fever with a few streptococci. Ehlers[4] was the first to call attention to its power of producing general infection. Brill and Libman,[5] in reporting a case of pyocyaneus bacillæmia, called attention to the great danger of post-mortem invasion, both by the bacillus pyocyaneus and the colon bacillus, due to their motility. According to these authors, there are only five cases worthy of credence, none having

[1] Zeitschrift f. Hygiene, vol. xv. p. 475.
[2] Gaz. Med., Nantes, 1895.
[3] Journal of Experimental Med., 1898, vol. iii.
[4] Hosp. Tid. Kjobenh, i. 1890.
[5] AMERICAN JOURNAL OF THE MEDICAL SCIENCES, Aug., 1899.

been reported previous to 1893, and they cite the belief of Schimmelbusch, that although local infections and resulting toxæmias were undoubtedly caused by it, yet it could not invade the organism.

The five cases considered by them as cases of general infection of this bacillus are as follows:

First, a case reported by Williams and Cameron in a child aged seven months. In this case no cultures were obtained from the blood ante-mortem, but post-mortem the capillaries of the liver, kidneys and spleen were found blocked with minute bacilli, proving by culture to be the bacillus pyocyaneus. The organs showed parenchymatous degeneration. Autopsy was made soon after death.

The second case, reported by Finkelstein, was a child, aged three months, with hemorrhagic diathesis. Blood cultures were made from the vein two days ante-mortem, showing the bacillus pyocyaneus and the colon bacillus. The presence of the bacillus pyocyaneus was determined in the stools and also in the blood aspirated one-half hour post-mortem. This is the first case in which the bacillus pyocyaneus has been found in the blood by culture during life.

The third case reported by Blum[1] from the clinic of Professor Escherich was that of a syphilitic child presenting the symptoms of fever, loss of weight, and also the presence of blood-filled vesicles on the great and second toes. Auscultation revealed a loud, rough systolic murmur. Death occurred after thirteen days. The post-mortem in this case showed a lobular pneumonia, the liver changes usually found with syphilis, pericardial effusion, and a thickening of the edges of the bicuspid valves. In this case the blood drawn from the ear on the day before death showed the presence of the bacillus pyocyaneus. The blood taken from the heart one and one half hour after death also showed the presence of the same bacillus by culture. Endocarditis was also experimentally produced in a rabbit.

The fourth case, reported by Czerney and Moser, a child, aged seven days. Blood cultures from the finger tips showed the bacillus coli communis and bacillus pyocyaneus. This case may be considered doubtful because of the method used. But many control experiments were made.

The authors report their case as the fifth, and claim it to be the second in which the bacillus was found during life in a manner excluding doubt. They also claim it as the first case reported in an adult, and thus show that the infection is not confined to infancy.

Bleeding from some portion of the body of the new-born child is not a very rare condition if we include the frequent cases of slight bleeding from the cord or the umbilical ulcer after separation. This form of hemorrhage is not associated with any general symptoms, unless the

[1] Centralblatt f. Bakteriol., Parisit. und Infections-Krankheit., February 10, 1899.

amount of blood lost be so great that a resulting acute anæmia be produced. If such be the result it means, in the vast majority of cases, that the medical attendant or nurse has been negligent, such hemorrhages occurring usually because of the patulosity of the umbilical vessels, which may be usually easily controlled by firm pressure and hæmostatics. The so-called precocious menstruation may be considered of the same type, as it is usually not associated with constitutional reaction.

In contradistinction, however, to this slight local bleeding we meet with a condition more rarely in which, as in the case reported, the bleeding is associated with general symptoms and the clinical picture is of an infectious process.

That this latter condition, to which the term hemorrhagic diathesis should be limited, is not very rare, is shown by the statistics of Ritter, who met with it 190 times in a series of 13,000 births at the Prague Lying-in-Asylum, and Epstein in the Prague Foundling Asylum, who pronounced it present in 8 per cent. in a series of 740 infants. Hecker, quoted by Osler, states its frequency as 1 in 500. Buhl's statistics are the same, while Spiegelberg gives it as 2 in 5000 and Genrich as 1 in 2800. The statistics of the Lying-in-Hospital in Boston are given as 0.57 per cent. in a series of 7225 cases.

Though authorities have agreed that the occurrence of hemorrhagic manifestations of a severe grade is less frequent in private than in hospital work, a fact which would certainly indicate the infectious nature of at least a portion if not all cases, still its causation until very recently has been attributed to syphilis or some local lesions, as ulcers of the intestinal tract. The comparative rarity, however, of any syphilitic manifestations in hemorrhagic cases, 2 per cent. to 6 per cent., according to Epstein, Ritter and Townsend, shows conclusively that it cannot be the cause in the vast majority of cases. Further, in Townsend's series of fifty cases, quoted by Rotch, there were in the nine cases examined by post-mortem no ulcerations of the mucous membranes or blood-vessel lesions found, and Duser,[1] quoted by Holt, found in a series of twenty-nine port-mortems but thirteen cases in which ulcerations of the mucous membranes were present, nine being in the stomach and four in the intestines.

Thus the relative infrequency of the occurrence of syphilis and ulcerative conditions in the cases of hemorrhage which have been reported may lead us to question their causal relationship even when present, and the doubt is still further increased when one remembers the statistics of Mracek, who found in a series of 132 cases of congenital syphilis but 14 per cent. the subject of hemorrhage.

[1] Thèse de Paris, 1889.

Ulcerations may, of course, be the cause of the hemorrhages in the few cases in which they are found, but authorities seem disposed at present to consider such cases as primarily septic, the ulcerations very probably being due to septic emboli predisposed to by the relation of the vessels of the stomach and duodenum, the thrombus being situated in the umbilical vein.[1]

Sepsis due to the ordinary pyogenic organisms seems at first sight, indeed, to play a more important causative rôle in the production of this diathesis than the previous conditions, since in an examination of 61 cases of sepsis Epstein found hemorrhagic conditions associated in 29, and Ritter in a series of 190 cases found the same association twenty-four times. That the relationship which, judged from the above statistics, might seem to exist between ordinary septic infection and hemorrhagic manifestations cannot be considered as valid is shown by the statistics of the Sloane Maternity Hospital, as follows: In a series of 1500 cases of labor in that institution during a period entirely free from septic trouble of any kind there was no case of hemorrhage observed, but in a subsequent series of 225 deliveries observed during the presence in the house of septic cases there were eight babies who developed hemorrhage. In the cases of hemorrhage observed, however, there were none of the usual septic manifestations, while in the cases of sepsis of the ordinary variety, present at the time, there was an entire absence of any hemorrhagic manifestations.

It would seem probable, therefore, that the etiologic rôle played by the usual type of pyogenic infection in the production of cases of hemorrhage can only be considered as one of predisposition.

As has been said, syphilis was formerly considered to be the cause of hemorrhagic disease, but the absence of syphilitic signs and history in many of the cases reported have led Neuman and Ritter and others to doubt the causal relationship and to relegate its influence in the production of these cases to that of predisposition. That syphilis has an important influence in predisposing to infection is well known, and has been emphasized by Epstein[2] and proven bacteriologically by Kassowitz and Hochsingen, and by Chotzen and Kolisko.[3]

Rotch thinks that the hemorrhages under discussion are probably symptomatic of different specific diseases, and the fact that the disease is self-limited in the cases that recover seems to point strongly to the same conclusion. It is a very interesting fact that cases after recovery show no tendency to a recurrence or to hemorrhage from the umbilicus on separation of the cord, even though it had been one of the bleeding-points during the course of the disease. Holt states also that circum-

[1] Landau, Ueber Melena der Neugeborenen, Breslau, 1874.
[2] Oesterr. Jahrbücher f. Pædiat., 1876. [3] Neumann, Archiv. f. Kinderheilkunde, 1890.

cision has been performed a few days after the cessation of bleeding without any resulting hemorrhage.

From the different cases of hemorrhage of this variety which I have found in the literature the general symptom-complex seems to agree well in general with the description given by Lartigau,[1] who says that the symptoms resemble other better known forms of septicæmia and some of the acute febrile diseases. He gives the symptoms at the outset as weakness, prostration and fever. Pulmonary disturbances and pleurisy are often present. Epistaxis is noticed at times, and tympany is present as a rule. Splenic enlargement he seems to consider quite constant. Diarrhœa and vomiting are present, and both at times contain blood. Cutaneous changes noticed are of the form of bullous eruptions or petechiæ, or rarely a papular rash may be observed. The eruption usually occurs on the trunk, limbs, or scrotum, and may appear either early or late. Death occurs in from forty-eight hours to five days as a rule, though sometimes later. Loss of weight is constant in cases which live long enough to show it.

The previous condition of the children who present this grave form of hemorrhage differs so widely that nothing can be learned by its consideration. Thus some cases before the development of bleeding have shown general weakness and intestinal disorders, and others, as was true in the present case, are perfectly healthy until the onset of the acute symptoms.

The situation of the hemorrhage may be in any tissue or organ of the body, but usually is found at the umbilicus or in the mucous membranes of the stomach or intestines or beneath the skin. Rarely internal hemorrhage may destroy the life of a child without any external evidences of bleeding. The onset is in the majority of cases sudden, and occurs usually before the twelfth day, though cases have been reported as late as the sixth week, and Lartigau,[2] in investigating an epidemic of dysentery due to the bacillus pyocyaneus, found three fatal cases in children aged nineteen months, five years, and four years respectively, and one fatal case in a man aged thirty-five years.

The cases that recover do so usually in a very few days, but no series of cases will present probably more than a third of the number resulting in cure. Townsend in a series of 709 cases found a mortality of 79 per cent.

The cause of death usually seems to be loss of vital force rather than an acute anæmia. But in the present case the latter seemed to me to be the cause of death, since the amount of blood lost was so great.

If we conclude, as seems to be justifiable from the symptoms presented by this child in connection with the post-mortem conditions and

[1] Brill and Libman, AMERICAN JOURNAL OF THE MEDICAL SCIENCES, Aug., 1899.
[2] Journal of Experimental Medicine, 1898, vol. iii.

bacteriological findings, that it was a true case of infection by the bacillus pyocyaneus, we have still to consider the question, to me the most interesting of any which the case presents, namely, how did infection occur in this instance without being operative in the other babies present in the wards of the hospital at that time and living under the same hygienic conditions? Unfortunately, the facts at command are not sufficient to form a basis for conclusions, and I can only present an inference which, while supported by absolutely no proof, still seems to me to be the most plausible explanation of the development of a sporadic case of this type.

In the consideration of cases of infection in general we can divide them all into two great groups: first, those in which the infection arises from internal causes, auto-infection, and, second, those arising from external causes. To consider the first group, or auto-infections: With regard to this particular case we may assume, I believe, that if the infection took place from within the source would be found in the intestinal canal, since the development of the most marked symptom, the hemorrhage, developed in that locality, and also since no collections of pus containing the bacillus in question were found which might in this case, as in several which have been reported, have served for the focus of infection.

In a somewhat hasty review of the literature of this bacillus I have been unable to find it as a usual inhabitant of the intestinal canal of children, and Kossel[1] says that among his many examinations of the bacteria of the normal and pathological intestinal canal he has never, except in one instance, found this organism. In the case in which he was able to determine its presence, that of a child, aged four weeks, the symptoms presented were those of diarrhœa, anorexia, and great weakness. The diapers, on exposure to air, became dark green, and the stools were dark green and slimy. The bacillus pyocyaneus was found in great numbers in the stools, with a great diminution of the usual intestinal bacteria. Green vomit developed later, and death took place thirteen days after admission to hospital. In this case pus was present in the right middle ear and the right nasal cavity. From it, and also from the secretions of the larynx and trachea, there were obtained pure cultures of the bacillus pyocyaneus. Kossel believed that the digestive disturbances which caused the child's death were due to this organism derived from the ear.

In this connection the stools of six healthy babies at the Maternity Hospital were examined for the presence of this bacillus, but with negative results, and while this is, of course, when taken alone, proof of nothing, still it is in accord with the investigations of Kossel just men-

[1] Zeitschrift f. Hygiene, xvi., 2.

tioned, and also has the support of Lartigau,[1] and of Calmette, whom he quotes. Booker[2] also says that its presence is very exceptional in pathological conditions of the digestive tract. Kranhalls[3] also speaks of its rarity in the same connection.

If, then, it may be assumed from the foregoing reports of various investigators that the bacillus pyocyaneus is not a normal inhabitant of the intestinal tract, infection must have arisen from without, from the air, hands of the attendants, or by means of the food and water. Prenatal infection through the placenta, as proposed by Neuman,[4] to explain cases of infection developing in previously healthy infants shortly after birth, cannot be considered as possible in the present case, the period between birth and the development of symptoms being too long.

While the air of the wards was not examined, it hardly seems probable that this was the source, as only this one child was affected, and also since Symmes in Bergmann's clinic was only able to find this bacillus present in the air once in a long series of examinations, and only then when there was much green pus present in the wards.

It seems to me hardly likely that the infection could have been carried by the hands of the doctors and nurses in view of the careful technique in vogue at the Maternity Hospital, and also because it is very improbable that this child would have been the only one to suffer had its infection had this origin.

As to food infection, the fact that the child was breast-fed would seem, in view of the well-known sterility of breast-milk, to render its relation as a causal agent impossible.

The usual sources of infection having thus apparently failed to explain the infection, I am in doubt as to its solution, and only offer the following inference as a possibility. It is a well-known fact that the occurrence of the bacillus pyocyaneus upon the skin and in the saliva is not very rare, the investigations of Mühsam resulting in its detection upon the skin of the axilla and in the inguinal regions in seven out of fifteen cases examined, while Pansinia and Frick[5] have also found it present in saliva and sputum, and Eberth (*Medicin. Centralblatt*, 1873, and Virchow's *Archiv.*, 1875, lxii.) determined it in sweat, as did also Audouard.[6]

Bearing the above-mentioned facts in mind it has occurred to me that possibly the explanation of the infection might have been found by bacteriological study of the nipple surface of the mother, both because the nipples form very good culture grounds if not carefully cared for, and because the aseptic condition in the puerperium depends more upon

[1] Journ. of Experimental Med., 1898, vol. iii.
[2] Johns Hopkins Hosp. Reports, vol. vi.
[3] Compt. rend. Soc. de biol., October, 1897.
[4] Archiv. f. Kinderheilkunde, 1890.
[5] Virchow's Archiv., cxxii., p. 424, and cxvi., p. 266.
[6] Journ. de Medicin de l'Ouest., 1879.

the intelligence of the woman herself than does any other portion of the aseptic technique. Moreover, it is to be remembered that among the class of women in the wards of our maternity hospitals the custom of moistening the nipple with saliva before suckling is not very uncommon, which fact, in the light of the investigations by Virchow and Audouard, may render my inference still more plausible.

In conclusion I must confess to a certain dissatisfaction in reporting this as a case of pyocyanic infection for the following reasons:

1. Because of the triple infection.

2. Because of the very localized area in which the bacillus pyocyaneus was found in comparison with the distribution of the other two organisms.

3. Because it was not detected in the alimentary tract, a situation where one might have expected to have found it because of the development of the dominant symptom in that locality.

4. Because I failed to attempt its detection in the blood during life.

In spite of the points just mentioned, which may seem to some to be of sufficient gravity to invalidate the case, I still believe it to have been a true case of infection by the bacillus pyocyaneus for the following reasons:

1. The negative history of the first sixteen days of life, followed by an acute illness, the symptoms of which can be explained only by the assumption of an infection.

2. The complete absence of any pathological lesion sufficient to cause death.

3. The presence of the bacillus pyocyaneus in the bile and liver tissue in a condition of virulence. As previously stated, I find that all investigators are in agreement as to the rarity of the occurrence of this organism within the body, and while future study may demonstrate its presence more frequently than we now suppose, I do not believe that it will be found to be a harmless inhabitant of the human body.

4. The association of hemorrhage as a symptom, the presence of the bacillus pyocyaneus having been established, seems to me to afford at least strong presumptive evidence that it was the dominant infection.

DISTORTION OF THE AORTA IN POTT'S DISEASE.

By Thomas Dwight, M.D., LL.D.,
Parkman Professor of Anatomy at Harvard University.

There appeared in The American Journal of the Medical Sciences for January, 1897, a description by me of a striking instance of this condition. The interest with which it was received by specialists

induces me to publish an account of two other cases met with in the dissecting-room in the season of 1899–1900. A further reason for doing so is that the case already published and one of the new ones present a greater distortion than I remember ever to have seen figured, and that the three together seem to explain the cause of the distortion and also hint at some of the dangers to which this condition exposes the circulation. I shall call the case already published A, and the new ones B and C.

The following is a brief recapitulation of the more important features of A:

The spine, that of a white man, aged fifty-one years, presented an extremely sharp angle at the junction of the thoracic and lumbar regions. The greater part of the lumbar vertebræ were fused with the lower dorsal ones. Consequently the spine was much shortened. The aorta descended, gradually crossing the column from left to right, to the head of the right ninth rib; then turned suddenly, making a long and almost horizontal curve with a deeper posterior concavity across the spine to the left, whence after another curve it reached the middle line a little above the promontory. Thus there may be said to have been three curves with two angles between them.

Case B. Male, white, aged seventy-five years. The spine presents a sharp bend at the junction of the thoracic and lumbar regions, with but little lateral or rotary distortion. The body of the tenth thoracic vertebra is somewhat compressed, being a little smaller than the one above it. Compression and distortion are more marked in the eleventh, the lower border of which almost touches the top of the body of the fourth lumbar. The bodies of four vertebræ, namely the twelfth thoracic, the first, second and third lumbars, have been practically destroyed, at least at the front, and the broken-down mass has solidified firmly. The fourth and fifth lumbar vertebræ are normal. As an effort at compensation after the cure the dorsal region above the lesion is slightly convex in front. The angle formed by two lines drawn through the centres of the bodies is about 70°. The prominence of the hump is made by the spine of the first lumbar. The sacrum consists of six pieces. The body of the first sacral vertebra is raised so as to bring the promontory above the line of the true pelvis. It is not impossible that this also is an effort at compensation for the loss of height. The sacrum is very long and flat, the coccyx being inclined strongly forward. The length of the spine in a straight line from the top of the atlas to the coccyx is 54 cm., and that to the promontory 40 cm. The average of the latter measurement in the male is probably from 60 to 63 cm. The length of the cervical region is 13 cm., the average being 13.3 cm., and that of the sacrum and coccyx 17.3 cm., both being measured along the anterior curve. The last measurement in specimen A is 17.5 cm. As this measurement is for most purposes a perfectly useless one, and extremely variable, I have not made enough observations to know the average, but I feel pretty safe in saying that it is not over 15 cm. Thus specimens A and B have this lengthening of the sacrum in common. The latter, so far as the spine is concerned, is much less interesting than the former. The angle is less acute, the destruction less, and the length greater. On the other hand

the distortion of the aorta seems to be even more remarkable. The vessel descends from the arch along the left of the fourth, fifth and sixth vertebræ. It then begins to incline to the left, crosses the head of the eighth rib and makes the first bend between the ninth and tenth ribs, the concavity being from 1 to 1.5 cm. beyond the spine. The aorta then runs forward, downward, and to the right, enlarging considerably. The convexity of the next bend is just to the right of the middle line opposite the seat of caries, between the bodies of the tenth thoracic above

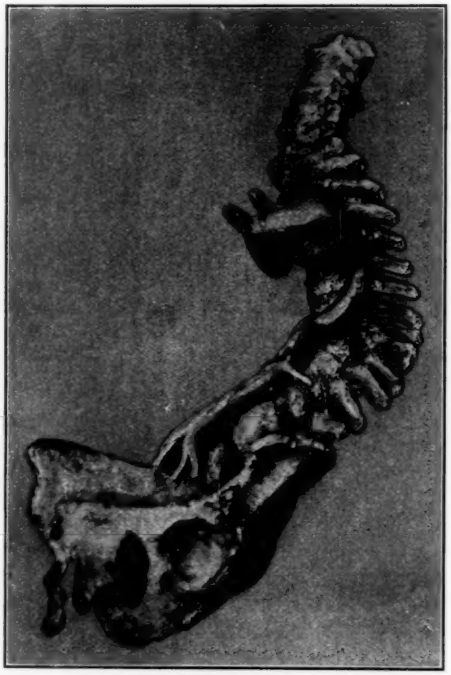
FIG. 1.

Case B.

and the fourth lumbar below. The entering angle of this bend on the left and behind is a very sharp one. The aorta then runs horizontally to the left, and at the same time a little backward along the top of the fourth lumbar vertebra. Near the back of the left lateral aspect of the spine it describes another bend, which is more gradual and graceful than the preceding ones. After this it runs again forward to the middle line, where it divides into the iliacs opposite the upper half of the fifth lumbar vertebra. Thus there are four curves with three angles. The right

iliac artery continues the course of the aorta, while the left one makes a sharp bend to the left, so that it may be called recurrent. The aorta is throughout remarkably large. A very important point is the egg-shaped enlargement of the transverse portion between the first and second bends. The cœliac axis springs from near the middle of this curve, and the superior mesenteric from just before its end. While the origin of these two large vessels might account for the diminution of the calibre of the aorta at the end of this curve, the cause of the dilatation at its beginning is not clear. The right renal artery arises from the con-

FIG. 2.



Case C.

vexity below the bend, and the left one a little sooner, just below the entering angle. (Neither of these shows in the figure.) The latter is so placed as to imply that the circulation of the left kidney might be carried on at a disadvantage. There is a moderate gradual diminution of the calibre of the aorta from this point.

CASE C. Male, white, aged fifty-nine years. The disease is in a higher part of the spine than in the others and the lesion less severe. The caries involves the lower part of the seventh thoracic and probably the upper part of the eleventh and all between these, so that there is

but about 1 cm. between these two vertebræ at the front. The prominence of the hump must have been made by the seventh, eighth, and ninth thoracic spines. There is a small excavation, not a part of the general destruction, on the left of the column, just in front of the head of the tenth rib. The coccyx is lost and the lower part of the sacrum injured, but the latter shows no sign of the elongation which is so striking in the other two specimens. The distance in a straight line from the top of the atlas to the promontory is 39 cm. The length of the neck is 12 cm., and that of the loins 15.3 cm. As the average of the latter is 19.9 this spine is presumably a small one, and though the distance from the atlas to the promontory is 1 cm. less than in B, I think that the loss of height is almost certainly less. The angle of the lines through the centres of the vertebræ is probably between 100° and 105°. The aorta presents a much less striking distortion than in A and B. The irregularity is limited to the thoracic region, and, indeed, does not involve the lower part of that. The arch is uncommonly high in relation to the spine, the horizontal part ending opposite the third thoracic vertebra. Thence it describes a loop to the left, lying on the heads and necks of the ribs, till at the head of the sixth rib it begins to sweep forward to reach the middle line at the eighth vertebra, just above the angle caused by the disease. It then turns downward and descends nearly straight, but with a very slight curve to the left, to divide into the iliacs near the middle line opposite the upper part of the fourth lumbar vertebra. The deviated portion in the thorax was but loosely attached to the thoracic walls, so that during the progress of the dissection great care had to be taken to retain the aorta in its original position, which was done by driving strong pins on either side of it at several places. The changes in calibre of the aorta are very curious. The early part of the arch is extremely large, probably owing to an exaggeration of the dilatation commonly seen after middle life. There is a gradual diminution throughout the arch, which is rather suddenly accentuated at its termination. Again, there is a rather sudden diminution in the lower part of the deviated portion, so that the descending aorta has lost much before the large abdominal vessels are given off. The abdominal aorta seems smaller than usual, so that there is a striking disproportion between the beginning and the end of the aorta.

Now to compare the three specimens. The injury to the spine is very much the greatest in A and the least in C. The last is the only one in which it is confined to the dorsal region. Both A and B have remarkably long and flat sacra. In A the flatness extends through the coccyx; in B the latter is thrown forward so as to give the combined regions something of the normal shape. In spite of the greater deformity of A the distortion of the aorta is greater in B, which has one curve and one angle more. In C the distortion of the vessel is entirely above the lesion. Lannelongue[1] teaches that the simplest form of distortion of the aorta is a simple bend corresponding with that of the spine, and apparently that the more complicated forms are caused by the pressure of the abscesses. That distortion may and does occur through such

[1] Tuberculose vertebrale, 1888, p. 104.

pressure is beyond question. A beautiful instance is figured by Lannelongue of a small abscess behind the aorta causing a sudden forward convexity of the vessel. But, if I follow him correctly, he implies that there is no other cause. Now in these cases, so far as I know, there were no abscesses, and even if one had been overlooked,[1] another more simple explanation is all-sufficient. It is that in case a number of the bodies of the vertebræ along which the aorta runs have been destroyed, the vessel can accommodate itself to the new conditions only by describing a series of folds. When the original relation between column and vessel has been lost, what else, so to speak, can the latter do? What determines the length and nature of each fold must of course vary with each case, with the degree and position of the lesion, and the point of least resistance. Thus in C the aorta is folded precisely as one would imagine it must be, with the exception that it is curious that the last part of the thoracic portion should have regained the proper position. I should have thought it more likely that it would only have reached the median line at the point of passage through the diaphragm. No doubt there was some restraining influence on the part just above it in this particular case that does not appear.

As to the size of the aorta: it is small throughout in A, large throughout in B, while in C it is large above and small below. In all three cases the aorta had been injected at its origin with starch mass, which, though not so distensive as some others, no doubt makes it abnormally large and acts particularly on the first part. Wishing to show the variations in the calibre of the aorta, I made a series of measurements of both B and C, but later discarded them. It is impossible that they should represent the original size, and unlikely that the distention should be equal throughout. Moreover, owing to the slow hardening of the mass, the vessel at times was more or less compressed in certain directions. Altogether it seemed to me more accurate to give general statements. The injection serves to make more evident the instances of dilatation of parts of the vessel, as for example, that at the first transverse bend of B, which are not easy to account for. On the other hand, injection probably tends to efface folds in the walls of the vessel.[2] The dangers to which the circulation in B is exposed have been alluded to. Were the aorta undistended some of the entering angles would probably be even sharper. It is well known that death

[1] I regret that I cannot speak with absolute certainty on this point. The fact is that when an important anomaly or pathological condition is observed in the dissecting-room the only thing to do for its proper description is to remove the body. But there are many reasons why this may be a very undesirable thing to do. If it be left, even with the best will of all concerned, the different points are rarely observed and noted at the proper time; and the proper time being passed they are often lost for good.

[2] The shadow in the figure of specimen C has exaggerated the depth of the bend below the deviated position.

is thought to occur from the sudden closure of the aorta at such bends. In view of the influence the condition of the aorta must exert on certain parts, and of the danger it may offer to life itself, these specimens are more than anatomical curiosities.

THE VALUE OF PEDICLED FLAPS IN INJURIES OF THE HAND.

By W. E. Schroeder, M.D.,
of Chicago, Ill.,
Associate Professor of Surgery, Northwestern University Medical School; Surgeon to Wesley Hospital; Surgeon to Provident Hospital.

The injuries met with in the hand which result in the destruction of the skin and subcutaneous tissues, leaving the tendons and all or some of the bones and joints intact, may in many instances be repaired by judicious treatment. These injuries are commonly produced by various kinds of machinery and by burns and frost-bites. It is not the purpose of this paper to discuss the etiology of such injuries in detail.

Amputation is frequently resorted to in contused lacerated wounds of the hand, whereas burns are usually allowed to get well of their own accord; that is, plastic operations are not often performed. It is for such cases that the pedicled flap method is to be recommended. No attempt will here be made to review the literature on other methods of skin-grafting, such as Thiersch's and the free flaps.

Elasticity and resistance are required of the skin in the palm of the hand, and neither one of these conditions is furnished by the Thiersch or free-flap method, although both of these methods may be used successfully on the dorsal surface of the hand. Enderlen, in his article, has shown that in Thiersch's graft the elastic fibres all die and that only a very thin layer of epithelial cells remains. It is also insufficient because of the ulcers that form when the hand is used. He also cites cases where free flaps were used and the microscopical examinations of specimens showed that parts of them died, resulting in the formation of connective tissue. Fenger permits me to report here a case of this kind. He placed a free flap over the metacarpal bones from which the fingers had been removed, and found that in time the flap shrank decidedly, and when the stump was used small blisters formed, resulting in ulcers. Microscopical examination showed a considerable increase in quantity of connective tissue. It finally became necessary to replace this with a pedicled flap (see Fig. 1), which terminated favorably. It was Dr. Fenger who, so far as I know, first applied the method which I used in the cases to be reported. I am also greatly indebted to him for the privilege of reporting two of his cases. His first case was

operated upon about ten years ago, and, as seen in the figure, the attempt was made to save the palm of the hand so that the thumb would have something to press against. This was entirely successful.

The second case was operated upon for an extensive burn of the palmar surface of the hand and all of the fingers but one. The result was very good.

Because of a series of photographs which I have had taken, showing each step of this operation as performed by Dr. Fenger, I report my last case in detail.

FIG. 1.

CASE I.—*History.* J. W., aged thirty years, colored; family history negative; physical examination negative. Patient was burned when he was six months old by accidentally getting his right hand into a grate fire. The hand remained untreated. When he presented himself I found the fingers in extreme flexion, the thumb in flexion and pronounced adduction, caused by cicatricial tissue. All of the joints were slightly movable and the tendons intact and not adherent. (See Fig. 2.) The function of the hand was almost entirely lost. All he was able to do was to hold small objects between the thumb and the nails of the first and second fingers. The development of the hand was somewhat arrested, the fingers being somewhat shorter and the hand slightly smaller than the left hand. There were no cicatricial ulcers present.

Preparation of Patient. The hand was prepared two days before the operation by cutting the nails, scrubbing with green soap and water,

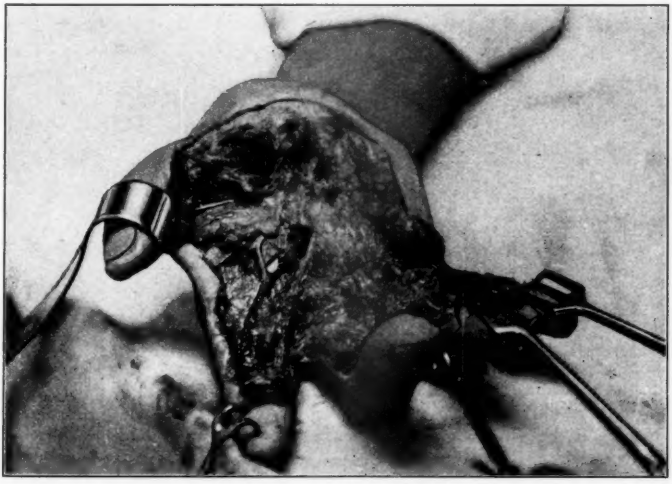
freely using alcohol, scrubbing with 1 : 1000 bichloride and then putting on 1 : 4000 wet bichloride dressings, which were changed every six hours. The skin over the right hip was prepared in a similar manner at the same time.

FIG. 2.

Before operation.

First Operation. 1. The cicatricial tissue was dissected off of the palm, fingers, and thumb. This left a wound extending from the carpo-metacarpal joint to the distal phalanges of the fingers and thumb. (See Fig. 3.) The deformity of the thumb was corrected, but the new posi-

FIG. 3.

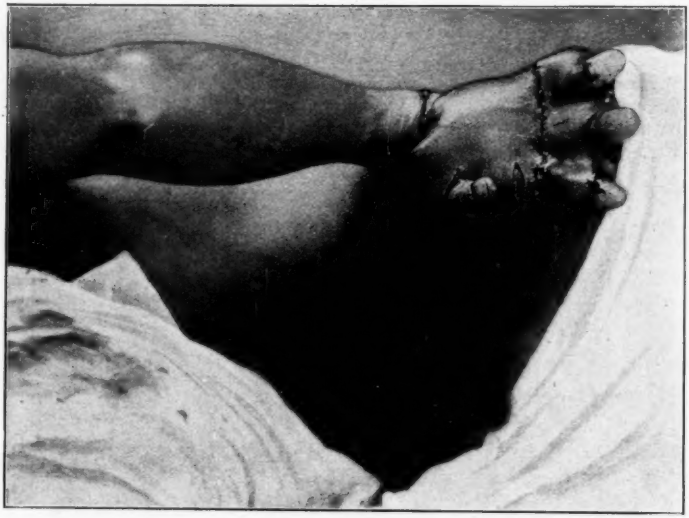


Cicatricial tissue removed.

tion was with difficulty retained. The first finger was flexed and held by the shortened anterior ligaments of the first interphalangeal joint, which were ruptured in extending the finger. The hand was now placed upon the hip and incisions made in the skin as guides. The upper flap was made wide enough to cover the denuded space above the first row of

digital furrows, having an anterior and posterior pedicle, the distal end of the thumb passing through the posterior pedicle. The anterior flap passes over to the crest of the ilium. The hand was now placed under this flap and the fingers separated and incisions made opposite the middle of the distal phalanx of each as guides. The hand was again removed and the pockets made, one for each finger, leaving attachments between the fingers for nourishment and better immobilization of the fingers. The hand was now placed in position and the upper and lower flaps united, as well as the lower border of the lower flap, to the fingers and the upper border of the upper flap to the edge of the skin of the wrist. (See Fig. 4.) There are several important precautions to be taken in this step of the operation, namely: 1. Not more than a quarter of an

FIG. 4.



Hand in place.

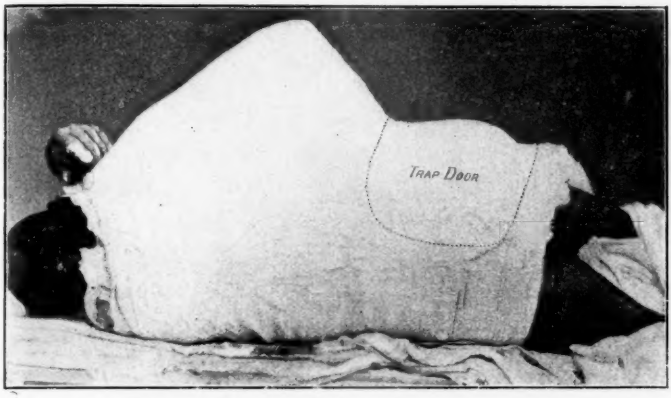
inch of subcutaneous tissue must be taken, because a thicker flap is clumsy and more difficult to unite to the skin of the hand. However, if more is taken, it will become absorbed in time. 2. There must be no tension on the pedicles. 3. The skin must have some subcutaneous tissue attached, or its vitality is endangered. 4. The edges of the skin of the hand must be undermined for at least one-quarter of an inch so as to allow of easy approximations.

Dressing. Sterile gauze is placed back of the hand and gauze drains back of the fingers. Next, a large quantity of gauze is placed over the hand, which is now in its place, and adhesive straps applied so as to hold it in place.

Plaster Cast. A plaster cast is applied, extending from the shoulder to the glutea-femoral fold.

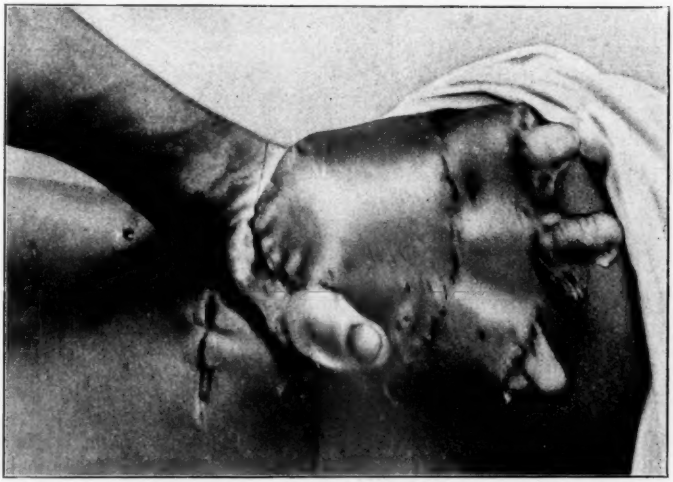
After-treatment. At the end of three days a trap-door is cut in the cast and the dressing changed. (See Fig. 5.) Boric acid solution is

FIG. 5.



Trap door.

FIG. 6.



Thumb liberated.

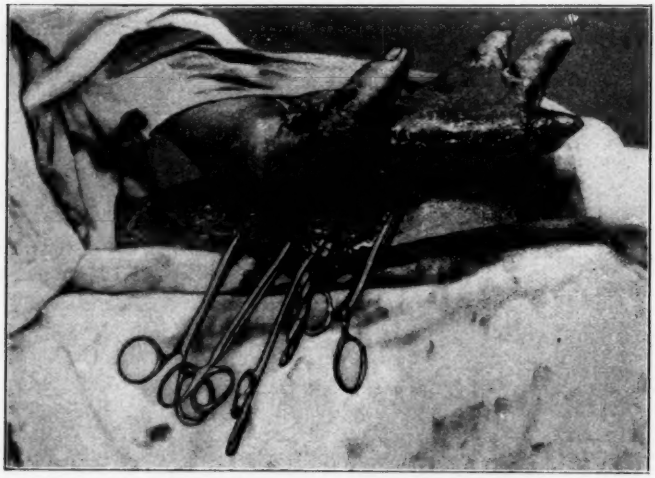
the strongest antiseptic used in these dressings. Gauze is reapplied and the door closed and held by a muslin bandage. This wound is dressed

every third day. The patient was able to sit up and walk about at the end of one week.

Second Operation. The second operation was performed on the eighth day, and consisted in dividing the inner pedicle to where the thumb protruded. Part of this pedicle was united to its former place. The flap was united to the thumb. (See Fig. 6.) A new cast was applied because of the broken condition of the old one. After-treatment the same as before.

Third Operation. On the sixteenth day the remainder of the posterior pedicle was divided and the flap stitched to the radial side of hand and index finger. Now, the flap is nourished from the anterior pedicle and interdigital septa and through the new adhesions formed. (See Fig. 7.) Dressing applied as before.

FIG. 7.



Medial flap divided.

Fourth Operation. On the twenty-second day the inner pedicle was divided and stitched to the ulnar side of the palm and little finger. The interdigital septa were divided, and thus the hand was liberated.

The wound in the hip was treated in the following manner:

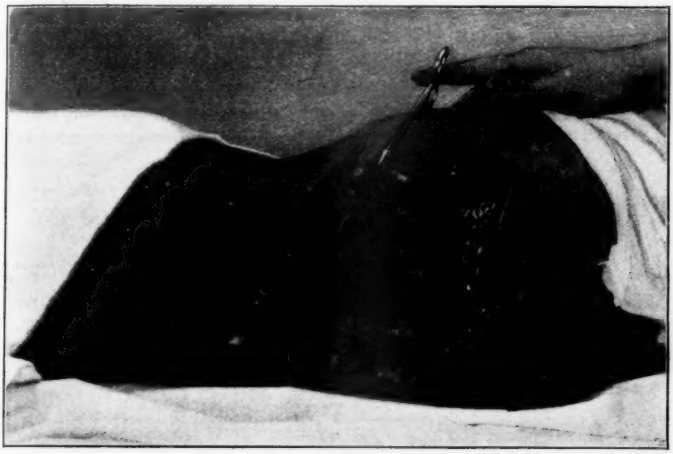
1. The granulations were removed with a sharp curette.

2. The skin was dissected back for a distance of two to three inches, and tension sutures and stitches applied so as to narrow the denuded space. The thigh, having been prepared the day before, furnished Thiersch grafts for the hip. Dressings applied, no cast. Dressings applied to the hand.

The Thiersch grafts took very nicely and on the eighth day the superficial epidermis, containing pigment, was shed, leaving them perfectly pale. The flap was reasonably thick and the subcutaneous tissue plus

granulations separated the fingers. (The wound in the hip before grafting is shown in Fig. 8.)

FIG. 8.



Wound on hip.

FIG. 9.

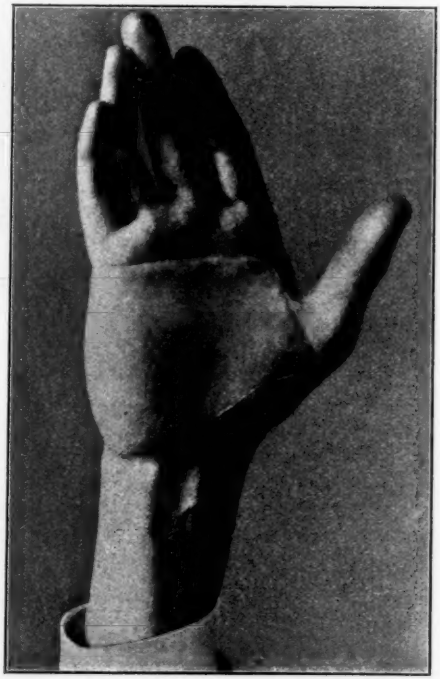
Hand after operation.

Fifth Operation. On the twenty-seventh day the bridges of flap between the index and second and between the little and ring fingers were divided and sutured to their respective edges of the finger.

Sixth Operation. On the thirty-second day I divided the bridge between the ring and second fingers, suturing as before.

Whenever an edge of flap was united to the edge of normal skin, it was necessary to dissect back the skin on the fingers or palm and freshen the edges and bevel those of the flap below, so that good coaptation

FIG. 10.



would be possible and primary union assured. This was done in all cases, and I also desire to state that it was not possible to avoid infection in any of my cases, but by frequent dressings and proper drainage this did not interfere with the final result, which was good. The joint opened in the index finger became ankylosed, as it did in the little finger of Case II. The usefulness of this hand has been very much improved, and the patient is now (three months after the operation) able to partially flex and extend the fingers. (See Fig. 9.) This was also the case with Fenger's patient. (Figs. 3, 4, 5 and 6.)

CASE II. The second case, which I operated on three years ago for an extensive burn of the palm and dorsum of the right hand produced by a hot mangle, gave a fair result. In this case the flexor tendons of the little finger were injured, and she would be better off with the finger removed. Otherwise, she has a useful hand, being able to do all ordinary work.

CASE III. I am indebted to Dr. Eskridge for this case. The patient, a young man, aged eighteen years, white, was caught between two rollers and suffered the loss of the greater part of the skin on the palmar surface and a portion of the dorsal surface of the hand. In the palm the flexor tendons were freely exposed. I operated upon this patient, assisted by

FIG. 11.

Dr. Eskridge, making a single flap from the hip. The free edge of the flap was united to the radial side of the thumb and upper and lower edge of the palm. Twelve days later Dr. Eskridge divided the pedicle and stitched the flap to the edge of the skin on the dorsum of the hand. A part of the flap on the dorsum died, and it became necessary to apply a Thiersch graft. (See Fig. 10.) This gave a most complete result. There is no limitation of motion and the boy's hand is as useful as ever.

CASE IV. This case is one which I saw in consultation with Dr. Eskridge, and I desire to thank her for the privilege of reporting the case. Patient, a young man, aged twenty years, white, was injured in a crushing machine, causing a severe crushing injury to the fingers of his right hand and a laceration of the skin of the palmar and dorsal surfaces of

the hand. The fingers died as did the flaps on the hand. This left the heads of the metacarpal bones and greater part of the palm and dorsum of the hand exposed. After removal of the dead tissues the doctor grafted the stump in the hip with a lower pedicle. After several weeks the pedicle was lengthened, cut low down, and the flap turned up on the dorsum and sutured. (See Fig. 11.) A small area had to be grafted with Thiersch grafts. The result was good, giving the patient a stump to press the thumb against.

Where one desires simply to cover the palm, dorsum of hand, or the palmar surface of the thumb, flaps may be taken with freedom from the chest or abdomen. Where the palmar surface of the fingers has been destroyed, it is much better to have double pedicles, and these can best be obtained from the hip. Furthermore, this position is reasonably comfortable. I would say, however, that in young and old patients such a procedure would be very trying and scarcely justifiable:

SUMMARY. Advantages of this method:

1. Mobility.
2. Elasticity.
3. Certainty of taking.

INTERNAL HYDROCEPHALUS FOLLOWING CEREBRO-SPINAL MENINGITIS.

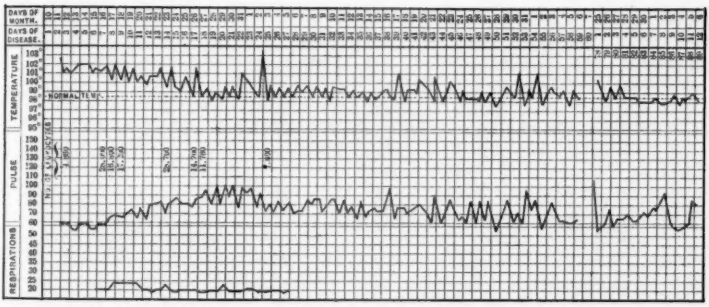
BY ELLIOTT P. JOSLIN, M.D.,
OF BOSTON.

THE following case of internal hydrocephalus occurring after cerebro-spinal meningitis is reported at the suggestion of Prof. Councilman and Prof. Mallory because the patient was under observation from the onset of the disease until its fatal issue, and because an autopsy was then performed. Through the courtesy of several other gentlemen, to whom I take this first opportunity of expressing my acknowledgments, an account of similar cases will be given, and following these a *résumé* of the literature, with a discussion of some of the pathological, but more especially the clinical, aspects of the subject.

CASE I.—Duration, eighty-nine days. R. C. was a young man, aged twenty-five years, with a good family history. He had had no illness except measles. July 10, 1899, he awoke in the morning with a headache, which became so severe at noon that he took to his bed. Later he vomited. The temperature was 102.4° F., the pulse 60 (see chart). I saw him for the first time July 12th. His temperature was then 101.4° F., pulse 60. Examination showed a medium-sized, undernourished young man, who was suffering comparatively little pain. Pupils, heart, lungs, and abdomen negative. July 13th, condition remained much the same. Widal and diazo reaction absent. Leucocytes, 4860.

Urine negative, except for a very slight trace of albumin. On the seventh day there was a leucocytosis of 28,000 and an abundance of fibrin in the freshly examined blood. Still more diagnostic was the stiff neck which appeared now for the first time. This was so pronounced that the whole body was raised if the head was lifted. Motion of the head sideways was also impaired. On the eighth day there was no headache, the neck was less stiff, and the leucocytosis had dropped to 17,200. In agreement with this was the temperature, which was now running at a degree's lower level, namely, 100° to 101° F. For the next five days it gradually, but irregularly, fell, and the pulse rose until, on the fourteenth day of the disease, it again reached 102° F., the pulse 80, and the leucocytosis 28,700. The following day it was noted that a watch could be heard only within six inches of the ears. No mastoid tenderness. Hyperalgesia of the fingers and toes was quite pronounced. Kernig's sign absent. The knee-jerks were slight, and the abdominal reflexes were now obtained for the first time. With the exception of a high temperature on the twenty-second to the twenty-fourth day, the patient's condition steadily improved for three weeks. He ate well,

CHART I.



was in good spirits, and had the merest trace of a headache. On the thirty-ninth day of his illness he vomited, and felt chilly and generally indisposed. His temperature rose once more to 101° F., and for two weeks it ranged between this point and the normal. Vomiting was repeated on the forty-second day, and again on the fifty-fourth, fifty-fifth, and fifty-seventh. Dr. E. M. Holmes kindly made an ocular examination on the fifty-eighth day, and reported normal, though rather pale, fundi.

As the patient had not improved for several weeks, he was moved into the country, where for sixteen days he gained steadily. During this time there was, with design, no record of pulse and temperature. He was seen daily, however, and it was thought that they rose but seldom above normal. About the seventy-fifth day he began to show less interest in what was taking place about him and said little. His appetite fell off and he vomited. Instead of going out-of-doors he kept his room. On the seventy-seventh day he was brighter, talked a good deal, and took a good amount of nourishment. The morning of the

seventy-eighth day found him as usual. He did have a "little time" vomiting, but this seemed transitory. Toward noon, however, he grew restless and thought he must pass water or have an evacuation, but when he got up little or nothing came, and he needed help to get back to bed. On one of these occasions he had a convulsion, during which he was partially conscious, but made incoherent remarks. Two hours after this attack he presented the typical picture of a cerebral anæmia. He was conscious, but his pallor was extreme. The pupils were equally widely dilated and responded only partially and sluggishly to light. No paralysis of any portion of the body was detected. The pulse was 108, regular, of fair volume, but low tension. Three hours later the pulse had fallen to 80, consciousness was completely lost, and he again became very restless. At 8 P.M. the pulse had dropped to 56. He was barely conscious and responded with "um," "ah" to questions. Toward morning the restlessness grew less. Dr. Walton saw him on this day and demonstrated that he was aphasic. Examination, including that of the eyes, was otherwise entirely negative save for a doubtful right-sided facial paralysis. By the eighty-second day, four days after the convulsion, the aphasia had disappeared. For the remaining nine days of his life he lay quiet in bed, spoke only when addressed, and was confused about well-known facts. Occasionally he was restless, complained of headache, and would moan in his sleep. He rarely vomited. At times the urine and stools were passed involuntarily. Rectal temperature was, as a rule, 98° F., and the pulse was about 60 to 70 and regular. He took comparatively little food, and, though he retained nutrient enemata well, gradually failed. On the eighty-eighth day there was in the evening a suspicion of Cheyne-Stokes respiration. By the following morning it was plainly present. He continued to grow weaker during the day, and toward evening almost imperceptibly died. The autopsy was performed by Dr. Mallory, who diagnosed an internal hydrocephalus on hearing the history of the case. His report is as follows:

Brain. Dura tense; convolutions flattened; vessels of pia moderately injected; pia slightly thickened in places along the course of the large bloodvessels; thickening more marked along the fissures of Sylvius and at the base of the brain, especially around the pons and medulla and in the neighborhood of the foramen of Majendie. The floor of the third ventricle bulges. On separating the two halves of the cerebrum the corpus callosum rounds upward. On opening the lateral ventricles they are found considerably dilated and filled with a clear, watery fluid, estimated at 70 to 100 c.c. The third and fourth ventricles also show a little dilatation. The ependyma of all four ventricles, but especially that of the fourth, is swollen and velvety to the touch; the normal markings in the floor of the fourth ventricle are obscured. Further section of the brain showed nothing remarkable.

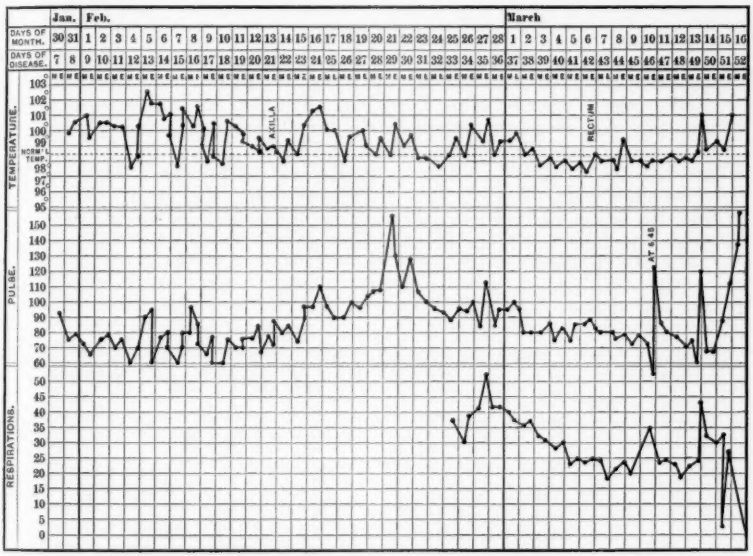
Anatomical Diagnosis. Internal hydrocephalus.

The case, then, in brief, is one of very mild cerebro-spinal meningitis which ran a fairly typical course for one month, with beginning convalescence; then came a month of stationary condition, during which there was slight pyrexia and an occasional attack of what was fairly typical cerebral vomiting. The first half of the third month showed steady improvement, which, however, terminated in threatening symptoms—apathy and vomiting—and these lasted three days; then came a convulsion with cerebral anæmia, from which the patient aroused

slightly, only to pass gradually into an apathetic state, in which he succumbed on the eighty-ninth day of the disease. That the hydrocephalus had existed for some time; that an increase of the fluid took place about two weeks before the patient died seems probable, and that the pronounced cerebral anæmia, which occurred on the seventy-eighth day of the disease, was due to the closure of the foramen of Majendie—all these suppositions seem consistent with the events recorded.

CASE II.—Duration fifty-two days. For the following record I am indebted to Dr. Edward E. Hooker, of Arlington. G. T. F., aged forty-three years, during an epidemic of influenza, was seized on January 23, 1897, with severe headache, photophobia, and backache, which extended to the hips. On January 30th the patient was in a mild,

CHART II.



muttering delirium, with hands picking at the bedclothes or holding the head as if in pain. Dejections involuntary during the next few days, and there was nausea and vomiting. On the twelfth day the temperature was subnormal, but it rose to 102.4° F. the next morning, when it was observed that there was slight twitching of the face and paralysis of the left levator palpebræ. On the sixteenth day the headache was worse, and the patient said his head felt swollen. Stupor, hiccough, nausea, vomiting, widely dilated pupils, partially closed eyes, and headache, together with involuntary dejections and a bladder requiring catheterization, constituted the picture on the nineteenth day. On the twenty-first day he began to moan, as if in agony, tossing and wringing his hands and rolling his eyes, and at times the thumb of his left hand turned in. The pulse rose to 140, and the hands were

cyanotic. This condition was temporary, however. For the most part of the time until the thirty-fifth day he lay in a semi-conscious state, though with almost constant moving of the feet. The thirty-fifth to the forty-sixth days were variable. For some hours he was very bright, during others delirious. Respiration was deep and occasionally sighing. Gradually the daily amount of food taken grew less; temperature and pulse fell from 100.2° to 97.8° F. and from 110 to 55 respectively, and the respirations, which were 55 on the evening of February 27th, dropped to 20 on the morning of March 9th. The forty-sixth day found him too dull to swallow properly. The pulse varied between 55 and 120 in three-quarters of an hour, and in a slightly longer interval of time the respirations fell from 40 to 24. From now on he steadily failed; no new symptoms appeared, but there was an intensification of the old. For the last few days of his illness there were marked irregularities in the cardiac and circulatory rhythm, which the chart brings out. On the day before his death Cheyne-Stokes phenomena appeared. On March 16th he died.

Autopsy by Dr. McGrath. Cranium: Sinuses normal; dura mater normal except for slight adhesion between it and the pia at the base of the brain. Brain: Weight, 1500 grammes. Removal necessitates separation of the slight adhesions above noted; the pia mater appears somewhat thickened over the base and upon the upper and lower surfaces of the cerebellum. Otherwise external aspect not remarkable. On section both lateral ventricles are distended with clear, colorless fluid, in amount about 150 c.c.; the ependyma seems somewhat thickened. On dissection the pia in the vicinity of the foramen of Majendie; is notably thickened. Otherwise the brain is not remarkable.

Case III.—Duration ninety to one hundred days. M. C., laborer, aged thirty-two years, single, entered the service of Dr. George G. Sears at the Boston City Hospital on October 18, 1899. Previous history not obtained; had worked until about August 1st, when one hot day he had a fainting spell. At home a week with headache and lassitude; then he was able to walk about, but a week later began to grow worse. Occasionally delirious, and the headache at times was extreme. For two weeks he was somewhat deaf. Though in bed a month, his appetite remains good and the bowels are "all right." Two weeks ago he vomited. Examination: Well developed and nourished. Left pupil reacts slightly to light, right does not; pulse 76, regular, fair volume and strength; heart and lungs negative; abdomen somewhat retracted and tense, but not tender; liver and spleen normal; knee-jerks and plantar reflexes present. Grasp of the right hand weaker than that of the left, and there is a corresponding diminution in power of the right leg, with ataxia on standing. Urine contains no albumin. October 21st, good appetite and sleeps well; no pain; leucocytes, 19,000. October 23d, Dr. E. E. Jack reported a violent neuritis (choked), with hemorrhages and patches of degeneration. October 29th, feels very weak; no pain; eats well. November 3d, leucocytes, 6900. November 4th, feels "awful sick;" much headache; otherwise no change in his condition until death, on the following day.

Autopsy by Dr. Pratt. Head: Calvarium thin, especially the left frontal and parietal bones, which are in places not over 2 mm. thick; the dura is not adherent, but is very tense; the gyri depressed and flattened out; sulci effaced. The pia-arachnoid space over the cortex

contains little or no fluid. The pia is somewhat opaque, and there are lines and collections of opaque white material along the vessels. This is apparently the remains of a purulent exudate, which has been replaced by connective tissue. The pia-arachnoid at the base is markedly thickened, and the pons, medulla, and cerebellum are bound together by a delicate network of fibrous tissue. Cover-slip preparation from the opaque material upon the cortex shows neither bacteria nor pus-cells. The interpeduncular space is so distended with fluid that it forms a distinct spherical cyst, projecting about 2 cm. above the surface of the brain when placed with the cortical side to the table. Lateral ventricles greatly distended with clear, pale fluid, 80 c.c. in amount. When the fluid escaped the cyst at the base collapsed. The fluid had a specific gravity of 1010, and contains one-twentieth of 1 per cent. of albumin. The foramina of Monro readily admit the little finger. The ependyma is smooth; the underlying cerebral substance is not softened; the choroid plexus is not swollen and is natural looking. The third ventricle and the aqueduct of Sylvius are markedly enlarged. The basal ganglia, particularly the corpora quadrigemina, are compressed. In the superior fovea of the fourth ventricle is a small, firm, yellowish-white opaque mass, several millimetres in size, that completely occludes the ventricle at this point. The inferior fovea is covered by a soft, reddish substance, apparently young connective tissue, that closes the foramen of Majendie and the apertures of Mierjejewski. Culture from the fourth ventricle sterile. Brain: Weight, 1340 grammes. The right middle ear is filled with thick, tenacious pus. Cover-slip preparation shows many polymorphonuclear leucocytes and numerous diplococci that stain by Gram's method. No other bacteria seen. Tympanic membrane and ossicles intact.

Anatomical Diagnosis. Chronic leptomeningitis. Chronic internal hydrocephalus, secondary to the occlusion of the fourth ventricle. Acute suppuration of the right middle ear. Atrophy of the bones of the skull.

Case IV.—J. E., aged fifty-eight years, married, born in Germany, and worked in a slaughter-house. On May 13, 1897, entered the service of Dr. Bowditch in the Boston City Hospital. Excessive beer drinker; "rheumatism" for nearly a year, and "sleeping spells." Two days before entrance drowsiness and stupor appeared, with pain in both ears, but without headache. On the day of admission, nausea. Examination: Well developed and nourished; semi-conscious, at times delirious; slight ptosis of the left eye; left pupil smaller than the right; neither react. The tongue protruded evenly; considerable tenderness exists below the mastoids. Pulse regular, of good strength and volume; arteries thickened and tortuous. Chest and abdomen negative; knee-jerk on the right, but left diminished. Cremasteric reflexes sluggish; urine normal; temperature 99.4° to 96.2° F.; pulse 65 to 90, and respirations 17 to 28. May 16th, the patient, at times delirious, but generally stupid, became unconscious and died May 19th.

Autopsy by Dr. Mallory. Brain: Dura about normal in thickness; pia much thickened over the whole brain, white and opaque; thickest and whitest along the large bloodvessels. It corresponds exactly with an acute inflammatory exudation in the meninges, except that it is white and opaque instead of yellowish. In its thickest portions the pia was exceedingly tough and could with difficulty be torn.

It stripped off from the brain-substance rather easily, and did not tear it much. Over the lateral portions of the brain the exudation was less marked, confined almost wholly to the neighborhood of the larger vessels. In places at the apex it was thick enough to obscure the brain tissue and the vessels beneath. In its meshes was a considerable amount of serum. At the base of the brain was an abundant whitish exudation, most marked over the medullary and upper portions of the cervical cord. The brain was removed with difficulty on account of its being necessary to cut through the adhesions between this portion of the cord and the foramen magnum. The optic nerves were swollen and soft and the commissure was shoved inward. Just back of it, surrounding the pedicle of the pituitary body, was a firm, grayish, translucent mass. The cranial nerves, other than the optics, seemed to be normal. There was little exudation over the pons or along the fissure of Sylvius. On opening the brain the lateral ventricles were found to be considerably dilated, containing each 30 to 40 c.c. of fluid. The ependyma was slightly granular. The bloodvessels were moderately injected. In the third ventricle was a small mass of whitish fibrin, attached at one end of the wall. In the fourth ventricle was a mass measuring 1.5 cm. in diameter and 3 mm. in thickness, unattached to the wall; its edges were rounded and extremely like a small piece of decolorized fibrin. The sides of the corpus striatum adjoining the third ventricle were swollen, firm, and transparent, and resembled closely the tissue described as lying just back of the optic commissure; on each side it was about 1.5 cm. in width. Outside of this on both sides, but particularly on the left, the central portion of the corpus striatum was very œdematous, soft, and transparent.

Anatomical Diagnosis. Organization of acute cerebro-spinal meningitis, with occlusion of the foramen of Majendie and the production of internal hydrocephalus.

Case V.—Duration sixty days. A. A. D., aged sixteen years, single, a brass and iron filer, entered the service of Dr. Vincent Y. Bowditch at the Boston City Hospital on May 2, 1898. As a child he had measles, and at four years scarlet fever, followed by "kidney trouble." Habits good. About the last of March he had a fight, was hit on the mouth and knocked backward against a wall, striking his head and neck. Present illness began two weeks before entrance, with languor and insomnia. While he was having these symptoms a man, with whom he lived up to February 2d, came to the hospital and died with cerebro-spinal meningitis. Three days before entrance he was suddenly seized with violent frontal headache, with dimness of vision. Worked until 6 P.M., when he vomited several times. The next day headache was more severe, and he had several chills and vomited. Dimness of vision increased; he staggered in walking, with a tendency to fall to the right. Toward night he became delirious. On the day before entrance delirium alternated with rational spells. Fell out of bed. A macular eruption appeared on the face, which disappeared within twenty-four hours. Pulse irregular and very slow. On the morning of entrance he had a convulsion; rigid, with hands clenched; cyanotic and stertorous respiration. Retraction of the neck was now for the first time noticed. Bowels constipated, but at no time involuntary defecation or urination. Examination: Well developed and nourished; stupid; flushed face; pain on pressure on the back of the neck, and slight

pain on movement of the retracted head. Right pupil larger than the left; both react. Conjunctivæ congested, and there is a questionable external strabismus. The tongue is covered with a moist, brownish coat; sordes about the teeth. Heart is normal except for prolongation of the first sound at the apex; pulse normal. Lungs and abdomen negative except for enlarged spleen. Knee-jerks lively. Small petechiæ over the skin. Leucocytes, 20,600. Urine normal. May 4th, slight ptosis of the left eye. May 5th, leucocytes, 9000. May 7th, steady improvement; no pain. May 8th, Dr. Lancaster reports a slight swelling of the optic nerve, with the veins considerably distended and tortuous; a few minute hemorrhages in retina near the disk; beginning optic neuritis. May 16th, patient doing well; no new symptoms except diplopia for distant objects. On May 20th he was discharged "well." The temperature did not rise above 99.4° F., and the pulse was for the most part of the time about 70, but was in the 50s on three occasions and just before discharge. June 17th, re-entry in the service of Dr. Henry Jackson. The patient had been comparatively well since leaving the hospital save for general bodily weakness, and on this account has attempted no physical exertion. Yesterday he felt so well that he rode a bicycle, but came home tired and with intense frontal headache. Went to bed and slept. About five o'clock this morning he got out of bed, walked ten feet, and had a convulsion, falling unconscious to the floor. At nine o'clock he had a second convulsion, at one o'clock a third, and at four o'clock a fourth, lasting about eight minutes, with frothing at the mouth. At 4.30 P.M., during another convulsion, he died. Examination made shortly before death showed him to be semi-conscious, delirious, opposed handling. Little, if any, stiffness of the neck; motions of the head resisted. Marked left external strabismus, with moderately dilated pupils, which react sluggishly to light. Pulse regular, full strength and volume. Heart, lungs, liver, and spleen negative. Knee-jerks lively. Leucocytes, 26,160. Temperature, 99° F.; pulse, 108; respiration, 29.

Autopsy. Brain weighed before opening 1650 grammes; after opening, 1530 grammes; 120 grammes of fluid. Dura tense. On stripping it back the surface of the brain was moderately injected; very dry. No thickening of the pia can be made out anywhere, unless possibly a little around the optic commissure and posterior to it. The floor of the third ventricle projects outward 3 mm. On opening the brain, the lateral ventricles are found to be distended and filled with a perfectly clear, watery fluid. No evidence of any inflammatory exudation can be found in the posterior cornua. The ependyma shows very slight granulation. The third ventricle contains a spherical, soft, yellowish cyst, measuring 13 mm. in diameter. It is adherent at several points to the choroid plexus of the third ventricle. Its wall is yellowish and to some extent translucent. On section the tumor is found to be encysted with a thin, delicate wall; the contents are grayish, rather opaque, and tenacious, sticking more or less together. Microscopical examination of this material shows it to consist partly of leucocytes, partly of large cells filled with fat-drops. The tumor mass obstructs the iter a tertio ad quartum. The wall of the third ventricle is thickened and granular. At the tip of the temporal lobe on the right side is a small, yellowish area, 2 mm. in diameter. Cyst due to puriform softening of the mass of fibrin in the third ventricle following cerebro-spinal menin-

gitis. Obstruction of the iter a tertio ad quartum. Internal hydrocephalus.

CASE VI. is not as typical as the others, but will serve as the connecting link between the acute cases and the chronic ones, in which the hydrocephalus fully developed. Duration forty days. R. P., an errand girl, aged seventeen years, born in Germany, entered the Boston City Hospital on October 21, 1898, in the service of Dr. George B. Shattuck. Family history negative. Usual children's diseases and diphtheria. Present illness began three weeks ago, with pain in the right arm, extending to the shoulder and down the back to the lumbar region. Intense pain in the frontal and apical region of the head, also pain and tenderness in the back of the neck. Considerable photophobia. For the first three days of sickness there were numbness and "pins and needles" in the legs and inability to move them. Chills nearly every night. In the chest is a feeling as of suppressed hiccoughs, with a slight hacking cough. Tonsils somewhat enlarged and painful when yawning. Micturition was accompanied by a burning, cutting sensation. Yesterday a temporary aphasic attack, lasting for ten minutes, and to-day another for five minutes. Vomited frequently at first, later irregularly. Examination: Well developed and nourished. Temperature, 100° F.; pulse, 90; respirations, 10. Pupils equal, dilated, and react. Neck painful to pressure. Heart and lungs normal. Spleen slightly enlarged. Reflexes normal. Leucocytes, 19,000. Widal reaction negative. On the day after entrance the patient had a severe chill. Mentality unimpaired, but in the afternoon a temporary paralysis of the right side of the face. October 24th, another chill, and headache so intense that she shrieked. October 26th, eyes and ears drawn to the left. Some difficulty in following the movement of the finger beyond the median line to the right. Constant twitching. Increased left patellar reflex and an ankle-clonus persisting for many minutes. A loud systolic murmur at the apex and over the whole cardiac area, which later disappeared. October 27th, clonus absent. October 31st, semi-conscious. November 4th, unconscious; temperature moderate; no more chills. November 8th, for the past two days temperature, pulse, and respiration all elevated; semi-retraction of the head; labored breathing. November 9th, died.

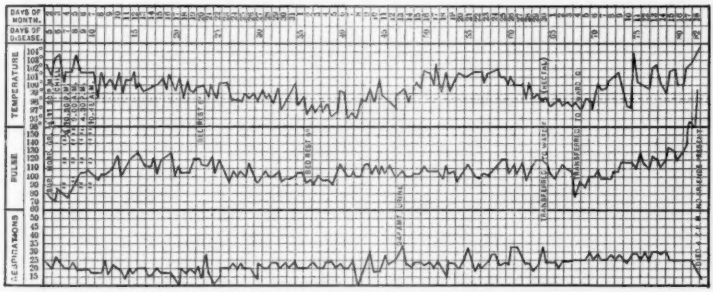
Autopsy by Dr. Mallory and Dr. Defendorf. Brain weighed 1295 grammes; dura rather tense. On removing it convolutions are a little flattened; vessels of the pia injected a little more than normal over the vertex of the brain; no thickening of the pia arachnoid; the olfactory bulbs are adherent to the skull. On removing the brain quite a little fluid escapes from around the base. On the under surface of the brain, over the circle of Willis and slightly involving the optic commissure, there is a whitish, rather translucent, tough deposit, varying from 2 to 3 mm. in thickness. A platinum needle could be drawn through it with difficulty. A little of a similar material is present over the vermiform process of the cerebellum and extending out toward the fissure of Sylvius. Otherwise than for this material the surface of the brain presents nothing abnormal. On opening the brain the lateral ventricles are somewhat distended with fluid, at least 20 c.c. each. The ependyma is everywhere finely granular. Over the corpus striatum the granules are decidedly prominent and larger than one ordinarily sees in this condition. In the right ventricle and in the inner posterior

corner of the corpus striatum, just where it joins the optic thalamus, is a small, yellowish, opaque elevation, 4 mm. in diameter and projecting 2 mm. above the ependymal surface. In the posterior cornua of both lateral ventricles is a soft mass of yellowish material, forming a little over 1 c.c. in each horn. Microscopically this contains more or less broken-down fibrin and a few pus-cells. The third ventricle is a little dilated and its ependymal surface decidedly granular. A similar condition is present in the fourth ventricle. In the lateral pockets is a little yellowish material similar to that in the horns of the lateral ventricle. The substance of the brain presents nothing abnormal microscopically.

Anatomical Diagnosis. Organizing meningitis. Granular ependymitis. Moderate degree of internal hydrocephalus.

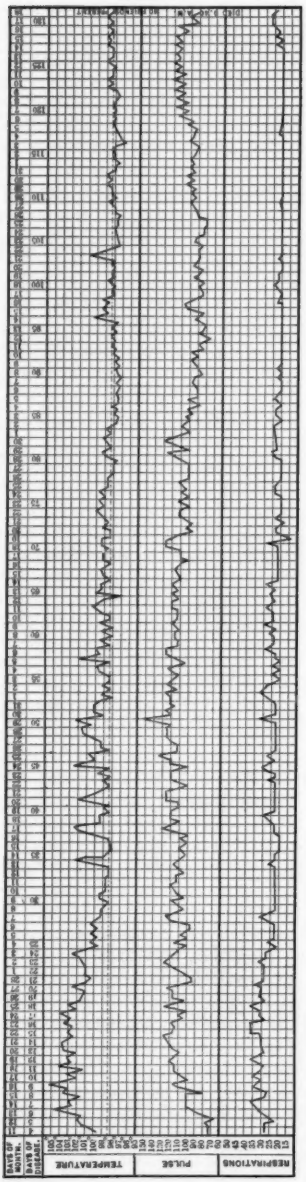
CASE VII.—Duration eighty-two days. Maggie D., aged twenty-four years, entered the service of Dr. W. W. Gannett at the Massachusetts General Hospital on October 2, 1897. Family history good. Two weeks before entrance she buried a child with cerebro-spinal meningitis. Four days before entrance she vomited without cause, and on

CHART III.



the following day was attacked with headache, backache, and vomiting, which again was of sudden onset, and, in conjunction with diarrhœa, caused great weakness. Examination: Well developed and nourished; good color, many herpetic vesicles about the lips. Pupils reacted, but were unequal, which the patient said was always the case. Neck rigid and tender. Chest and abdomen normal. Knee-jerks and plantar reflexes present. Leucocytes, 23,600. Diplococcus intracellularis Weichselbaum was grown from the nasal discharge. Vomiting and headache continued after entrance. The pulse was irregular, and fresh crops of herpes appeared. Between the seventh and twelfth days she improved, but occasionally had diplopia. Many hyperæmic papules appeared on the thighs and legs, which later changed to pustules. During the next week she was better and worse by turns. At times the heart was weak and the respiration suggestive of the Cheyne-Stokes type. Leucocytes on the nineteenth day, 10,600. On the twenty-fifth day the eyes were examined by Dr. Cheney: Slight congestion of the retinal veins. Persistent vomiting, with resulting emaciation. Leucocytes on the forty-eighth day, 9600; on the fifty-fourth day, 8200; on the sixtieth day, 7000; and on the seventieth day, 16,200. The left pupil lost its reaction

CHART IV.



to light on the eighty-first day, which was the day of her death. For some days previous the knee-jerks had not been obtained. Vomiting, however, ceased during the last week of life. Cheyne-Stokes respiration at times manifest.

Autopsy. Meninges of convexity dry, not specially injected, and showing no exudate. Convolutions somewhat flattened. At the base over the pons and a portion of the medulla the meninges were infiltrated, with a yellowish, semi-translucent, somewhat jelly-like material. The region of the chiasma was free from exudate. On removing the brain a stream of clear, watery, colorless fluid squirted from an opening in the neighborhood of the optic chiasma. Lateral ventricles and fourth ventricle markedly dilated, and contain a rather cloudy fluid. In the lateral ventricles some large, fibrinous flocculi were present. Dissection of the brain shows nothing remarkable except that the tissue of the corpus callosum and fornix posteriorly is perhaps softened. Lateral sinuses free. Cord: No accumulation of fluid in the subdural space. In the cauda equina and in the meshes of the pia of the thoracic and lumbar portions are accumulations of opaque, yellow exudate arranged in patches. Exudation is not extensive. In the thoracic portion the cord seems softer than normal; this may be due to injury in removal.

Anatomical Diagnosis. Subacute cerebro-spinal meningitis; internal hydrocephalus.

CASE VIII.—Duration one hundred and thirty-one days. (Chart IV.) J. C. entered the service of Dr. R. H. Fitz at the Massachusetts General Hospital on February 11, 1898. Parents and one sister died of phthisis, and another sister has the disease. Except for bronchitis at three years of age he has been well. February 8th he was exposed to cold and wet, and felt chilly, had pains in his arms during the night, and was delirious.

February 9th, headache and vomiting commenced and persisted. Examination: Fairly developed and nourished man, in much pain. Pupils small; reaction sluggish. Neck and back stiff. At the apex and base of the heart a faint systolic murmur. Knee-jerks present. Leucocytes, 36,000. Urine slight, trace of albumin, rarely a hyaline cast; no diazo reaction. During the next few days delirious and in pain. Leucocytes, 12,700 on the sixth day, but 20,000 on the eighth. On the following day he developed a pneumonia of the right lower lobe, with a leucocytosis of 34,000. Four days later, still very ill, and leucocytes 40,300. On the seventeenth day a patch of pneumonia appeared in. the left back, and the white corpuscles were 50,800. For the next seventeen days he continued to improve, but the white count fell only to 26,000. By the fifty-ninth day a living skeleton; answers "yes" and "no" and "fine;" lies with his eyes closed and arms folded Micturition involuntary and vomiting frequent. On the twenty-fifth day he had a chill, and leucocytes 45,700. No plasmodia. A bacillus similar to the tubercle bacillus was found in the sputum, but pronounced by Drs. Ernst and Wright not to be the tubercle bacillus. From about the sixtieth day on the temperature was usually normal or subnormal. At times there appeared to be some slight improvement in his condition, but on the one hundred and sixth day his weight was 63 pounds instead of 130 pounds as at entrance. Leucocytes still increased, being 26,000. On the one hundred and twenty-second day, failing slowly and more emaciated. Skin dark; bloodvessels distended; bedsores on the back, thighs, legs, knees, ankles, elbows, and shoulders, and there seemed to be only just enough tissue left to hold the bloodvessels together. Cheyne-Stokes respiration ushered in the end on the one hundred and thirty-first day.

Autopsy. Chronic leptomeningitis of the base of the brain and of the spinal cord, with hydrops ventriculorum and softening of the right lenticular nucleus. Head: Dura normal; pia of convexity normal; convolutions flattened. At the base an accumulation of thin, cloudy fluid. The pia at the base shows fibrous shreds and some thickening. Frontal lobes more adherent than normal. Lateral ventricles considerably dilated. A large part of the right lenticular nucleus mushy, soft, and brown in color. Vessels and cerebral tissue otherwise not remarkable. Middle ears normal. Spinal cord: Dura everywhere rather firmly adherent to the pia-arachnoid by translucent tissue. Cervical cord fluctuating to the touch. On section of the dorsal and lumbar cord the nerve tissue seems to show extensive softening, is soft and mushy. No fluid accumulation in the dural sac. The cervical region seems somewhat enlarged and fluctuant.

A Résumé of the Literature on Internal Hydrocephalus following Cerebro-spinal Meningitis.

Internal hydrocephalus has been known to be a result of epidemic cerebro-spinal meningitis since 1805, the year in which the disease is usually considered first to have appeared.

In the same year[1] (1805) a case occurred at Strasburg, terminating fatally after two relapses, the last one being accompanied by cerebral

[1] Quoted by Webber. Boston Medical and Surgical Journal, vol. lxxv., p. 85.

symptoms. At the post-mortem the vessels of the head and membranes were engorged with blood; serous fluid was found between the meninges above the hemispheres. The brain was soft, the ventricles were dilated and contained about four ounces of turbid serum, with a deposit of pultaceous, puriform matter lining their cavity. (*Journ. de Med., Chir. et Phar.*, etc., vol. x.)

Foerster,[1] in 1863, gave a very good description of this complication. He says the inflammation usually runs a fatal course, a recovery being seldom complete; sometimes the acute symptoms cease and the case becomes chronic. Meanwhile the secretion of serum continues and forms a chronic hydrocephalus. The ependyma at times remains forever generally or partially thickened.

Ziemssen and Hess[2] published a long article upon "Clinical Observations in Epidemic Cerebro-spinal Meningitis," in which they record four cases of long duration which showed at the autopsy internal hydrocephalus. No decision was reached as to whether the process was inflammatory or due to a passive congestion from pressure on the veins of the choroid plexus.

To this excellent account Ziemssen,[3] in 1874, added a more extensive description, which to-day embodies more than any other article on the subject. He says that lasting alterations in the brain, the spinal cord, and their membranes are not by any means seldom. Among these chronic hydrocephalus appears to form far and away the most frequent after disease. He explains this change as follows: The slightly altered, perhaps thicker, exudation is absorbed slowly or quickly in the second week, through fatty degeneration of the cells and fibrin, or, in consequence of absorption not occurring, shrivels up into a caseous mass. While the connective tissue of the soft membranes proliferates the hyperæmia of the brain-substance subsides and the purulent effusion in the ventricles increases. In the twenty-seventh or twenty-eighth week the soft membranes show a pulpy hyperplasia or already cicatricial thickening; the caseous remains of the exudate contract still further; the effusions in the ventricles become more moderate in amount, but quite clear, while the cellular elements in the dependent parts of the ventricles are thickened to small, caseous plates. The earlier hyperæmia of the brain is completely gone; the brain is anæmic, even œdematous; the ependyma of the ventricle is thickened and clearly granulated, and the choroid plexuses bloodless. The diminution of the brain substance depends on the degree of the hydrocephalus, but can be very considerable. It almost appears as if the increase of the ventricular effusion was conditioned in the first instance by the contractions and thicken-

[1] Path. Anat., 1863, p. 597.
[2] Deutsches Archiv., 1865, vol. i.
[3] Handbuch d. acuten Infect. krankheit., Zweiter Theil, 1874, p. 683.

ings of the soft membranes. At least the usual stage of apparently progressive convalescence between the acute attack of meningitis and the appearance of the hydrocephalic signs speaks for this view.

The symptoms of the secondary hydrocephalus consist of paroxysms of severe headache, and also of pains in the neck and extremities, with vomiting, unconsciousness, convulsions, involuntary evacuations of feces and urine.

These attacks recur either in a pretty regular type or make pauses which not rarely last for weeks, and during which the activity of the mind and senses may be unimpaired, the appetite excellent, the sleep restful, and the patient in good spirits and gaining strength. If these interims last for a considerable time we easily fall into the error of supposing that the disease has taken a favorable turn, until a new attack dispels the illusion. Generally, however, the condition during the interval is not so favorable that such a prognostic error is possible; there persists the general hyperæsthesia, increased reflexes, furthermore slight contractures or pareses of the extremities, psychical disturbances, and other symptoms.

Of great practical importance is the question whether and to what degree these hydrocephalic effusions are capable of retrogressive changes; in other words, is cure of a secondary hydrocephalus ever possible? Generally it is not; "yet I have seen some cases in which a complete, and others in which an incomplete, recovery took place."[1]

In the interval between Ziemssen's two publications Collins[2] gave an excellent report upon "Epidemic Cerebro-spinal Meningitis." He says a return of this symptom (pain in the head) in company with vomiting and convulsions but too frequently points to the issue of the disease in hydrocephalus. He records the case of a boy, aged ten years, who died on the sixty-sixth day of his illness, with, he thinks, the cause of death due to an occlusion of the cerebro-spinal opening. This is the first direct mention of this as the cause of the hydrocephalus in such a case that I have been able to find.

In 1876, Neville Hart[3] recorded four or five typical cases of hydrocephalus following meningitis, and in describing the symptoms it is interesting to note that dilatation of the pupils was constant in the late stages. The respiration was irregular, there was great emaciation, bedsores, paralysis of the bladder and rectum, and in a few cases of the pharynx.

Heubner[4] describes this type of cerebro-spinal meningitis. He says that hydrocephalus is to be recognized by paroxysms of pain, vomiting,

[1] Archiv f. klin. Med., Band i., p. 389.
[2] Dublin Quarterly Journal, 1868, vol. xlvi., p. 170.
[3] St. Bartholomew's Hospital Reports.
[4] Eulenberg's Encyclopædia, third edition, pp. 430, 435, 437.

coma, and convulsions. The weakness of memory, intelligence, and speech may be due to the same cause.

Eichhorst[1] says he has repeatedly seen hydrocephalus develop, and in two cases far advanced in convalescence there suddenly appeared headache and apathy, which resulted in a few hours in death, for which the only cause was a marked internal hydrocephalus.

Von Leyden[2] calls attention to epileptic convulsions as characteristic of meningitis of the convexity, but to coma and cerebral paralysis as significant of large exudations in the ventricles. Later,[3] he says the danger of the disease lies more in the danger to the brain-substance through the exudate than through the severity of the infection.

This by no means exhausts the literature, but most of the articles written on the subject are quoted under one or another of the above references or are referred to on page 128 of the *Report of the State Board of Health of Massachusetts*, 1898, which was written by Councilman, Mallory, and Wright.

PATHOLOGY. A comparison of the reports in our eight cases of cerebro-spinal meningitis shows a marked uniformity. In all save one the hydrocephalic fluid was clear and colorless, and was sterile in two instances in which it was examined; in a third, diplococci were found, though not the specific diplococci of the disease under discussion. (The presence of bacteria in this case (VII.) may account for the peculiarity of the high temperature which existed in the latter weeks of the illness and rendered the case unique in this respect—*i. e.*, a secondary infection may have taken place. This is by no means certain, however, as the bacteria were few in number.)

That the hydrocephalic fluid was sterile we should expect, for it is an acknowledged fact that the bacteria in these cases disappear in the early part of the disease. Akin to this is the absence of collections of pus, save in the cases of shortest duration (VI. and VII.), in which leucocytes were found in the cornua of both lateral ventricles.

The pia is reported thickened in all cases in the region of the roof of the fourth ventricle, and in most of the cases elsewhere as well, particularly at the base of the brain. The thickening was of marked degree, in one case resisting the thrust of a platinum needle, in another necessitating a division of the adhesions. This thickening is due to organization—that is, to the formation of granulation tissue, which starts from the pia and grows into the fibrinous exudation, gradually dissolving and removing it. It covers enough surface not only to bar the foramen of Majendie and the two lateral openings of Mierjejewski, but to render impervious the whole roof of the ventricle.

[1] Hand. der spec. Path. u. Therapie, 1897, p. 527.
[2] Zeitschrift für klin. Med., 1887.
[3] Nothnagel Series, vol. x., p. 291.

There does not appear to have been any thickening of note in the above cases about the bloodvessels except in Cases III. and IV., nor were the choroid plexuses injured.

The changes in the brain resulting from the increased pressure were not different from those of other cases of hydrocephalus. The flattening of the convolutions varied with the amount of fluid. The bloodvessels of the pia were, as a rule, injected.

There is less uniformity in the condition of the ependyma. In the first case it was swollen and velvety, in the second somewhat thickened, smooth in the third, and very slightly granular in the fourth, fifth, and sixth. Nothing abnormal was observed in the seventh and eighth.

In the light of the records of the preceding cases and the observations collected from other sources, a very definite description of this sequel of meningitis can be given.

Unless the disease is rapidly fatal, toward the end of the second week the intense hyperæmia of the soft membranes subsides, and the fibrinopurulent exudate begins to undergo absorption in the same manner as in any other portion of the body. At the same time the large or small effusion in the ventricles, which is turbid with flakes of pus in the extremities of the cornua, changes its character. This purulent material is either absorbed, owing to the abundant vascular supply furnished by the choroid plexuses, or shrinks into caseous masses which are deposited on the walls of the ventricle. In either event, by the end of the third week, if not before, the fluid in the ventricles and subarachnoid spaces is clear, and, as experience has recently shown, is sterile. This latter fact is of especial importance, because it helps to exclude the idea of a chronic inflammation of the ependyma as a cause of this hydrocephalus.

During the next few weeks the organization of the exudate occurs, and in our opinion, according to the extent of this process of repair, which in turn is necessarily dependent on the location and amount of the exudate at the commencement of the disease, rests the final outcome of the case. If the nerve-roots have been embedded in the exudate they are liable to suffer from the cicatricial thickening about them. If the inflammation has been extensive and the meninges of the base of the brain "plastered" in the region of the roof of the fourth ventricle, the foramen of Majendie and the neighboring foramina of Mierjejewski may be obliterated and the velum interpositum thus rendered impervious. We have taken for granted that there is such a foramen and such neighboring foramina, as this appears to be the prevailing opinion; but should one not accept this supposition it would not alter our argument, for the whole velum is often covered with the exudate, and the whole might be easily rendered impervious. A hydrocephalus would then be the natural result.

In 1863, Hilton, in his *Lectures on Rest and Pain*, emphasized this factor in the etiology of hydrocephalus. He mentions and delineates the specimen from a case which he autopsied in 1844. Collins followed Hilton's lead, and in a case of cerebro-spinal meningitis of his own attributes the resulting hydrocephalus to the closure of the foramen.

But by no means is this the only method for the production of an intraventricular effusion non-inflammatory in character. In many cases the lateral and third ventricles are dilated, but the fourth not. In Case V. of our series this occurred, and the cause was manifest — there was an obstruction of the iter a tertio ad quartum. This is not, however, a unique occurrence, and it is only to be wondered at that this does not occur more frequently, for it is so easy for a collection of fibrin to become organized at this spot. Similarly the foramina of Monro may be plugged, one or both, and such would seem to be the case in those ordinary forms of chronic hydrocephalus in children in which puncture of one ventricle serves to drain only that side of the brain. Naturally there is no reason why the veins of the choroid plexuses should not be compressed by the cicatricial contractions; but it is not clear how this occurrence alone would explain the hydrocephalus, providing the paths for the circulation of the cerebro-spinal fluid were intact. It must be a complete obstruction of the veins, indeed, that would suffice to produce such an increase in the amount of cerebro-spinal fluid as to start in operation that vicious circle hydrocephalus leading to compression of the brain and its vessels, thereby preventing the removal of the excess of fluid and at the same time favoring an increase in the hydrocephalus.

Symptomatology and Diagnosis. It is impossible to decide definitely when the hydrocephalus begins in these cases, because the symptoms of meningitis and hydrocephalus are in the first instance so similar, and in the second place both conditions may exist together from the start. In three cases (I., III., and V.) an abatement of symptoms or an interim of apparent convalescence followed the first attack, while in four patients (II., VI., VII., and VIII.) the active stage of the meningitis merged into a condition which was evidently directly attributable to the hydrocephalus. Case V., for example, so far recovered from his initial attack that he was able to take a bicycle ride, though with disastrous sequences. Case I., with some help, made his way up and down stairs. Case VII., however, was of eighty-one days' duration, but presented no symptoms of hydrocephalus in the last month other than the ravages upon the system of a long illness, which were not present in the first four weeks of the disease.

These cases further show that no relation exists between the development of the hydrocephalus and the severity of the original attack. Cases I., III., and probably IV., were of mild character, while in the

others the disease was severe. From this we may infer that it is not so much the extent and intensity of the initial inflammation as it is the location of the same.

Analysis of Symptoms. Apathy or mental lassitude on the part of the patients is the predominant factor in the symptomatology. The histories lead us to believe that this was present in each case which was under observation. Beginning with simply apparent drowsiness and indifference, it gradually increases until the patient appears asleep the greater part of the time. In reality he is not, for he is easily aroused and responds to a question, but only to drop off again into his former condition. One can trace the gradual increase of this lethargy in the steady diminution of voluntary efforts and remarks on the patient's part. He is profoundly passive. This state may pass into one of unconsciousness, as in Case II., and the patient remain in this condition until death. Usually this is temporary, and the patient finally dies somnolent rather than unconscious.

Vomiting took place in all but one of the eight cases. Its irregular occurrence, the absence of any relation to diet or treatment, and the lack of other gastro-intestinal symptoms stamped it as of nervous origin.

Headache was severe in one-half of the cases, but in three of the others it was distinctly of mild type, and in one patient was wholly absent. The somnolence of the patients may have masked its character, but it is certain that it was far less severe than the headache of onset.

The pupils were dilated or reacted sluggishly or not at all in seven of the patients. In the final case no note is made of their condition.

Muscular weakness and emaciation were the rule, but not out of great proportion to the length of the disease and the small diet.

The temperature curves are extremely suggestive, because they were of normal or subnormal character for much of the illness, especially for the few weeks before death. In Case II. there was a rise for about forty-eight hours before the end, and in Case VII. the curve is so similar to that of typhoid fever that we are glad to know its absence was confirmed by the autopsy. The relation of temperature to pulse is most striking. In Cases I., II., VI., and VII. the pulse repeatedly *rises* as the temperature falls, and *vice versa*. This reverse of the rule is not absolute, and holds chiefly for the early part of the disease. Toward the end the pulse falls, and then appear those great irregularities in rate from hour to hour which have always impressed the physician. These later irregularities, however, as well as the Cheyne-Stokes phenomenon, which occurred four times, are terminal manifestations.

Quite in accord with the pathological findings is the most striking clinical feature of these cases, namely, their long course. The duration of the various cases was successively eighty-nine, fifty-two, ninety

to one hundred (?), sixty, forty, eighty-two, and one hundred and thirty-one days.

Mental apathy, vomiting, headache, dilated or sluggish pupils, with a normal or subnormal temperature when they occur a month or more after the onset of cerebro-spinal meningitis, point to hydrocephalus. Convulsions, paralyses, including aphasia, optic neuritis, dizziness, involuntary passage of excreta, and bed-sores may be concomitants, but they are less common and not as distinctive. With the absence of fever in these cases it is surprising that in Case III., five days before death, the leucocytosis was 19,000; in Case V., on the day of death, 26,000; in Case VIII., with an almost unbroken normal or subnormal temperature for weeks before and after, that it should be on the ninety-fifth day of the disease 26,000.

PROGNOSIS. Von Ziemssen[1] says he has seen some cases in which a complete, and others in which an incomplete, recovery took place. Hart[2] mentions two cases of recovery of the chronic form. Of course, in the nature of the case, positive data regarding recovery from the hydrocephalus are and must be uncertain, because of the difficulty of the diagnosis. In looking over the 111 cases recorded in the *Report of the State Board of Health of Massachusetts* I find that twenty-two cases were of forty or more days' duration. The mortality for the whole number of cases was 68 per cent., but of the twenty-two mentioned was 23 per cent. Of the seventeen cases which eventually recovered eleven had some definite signs of hydrocephalus, four evidently did not have hydrocephalus, while a mastoid abscess delayed the recovery of one case, and the status of the remaining patient was not determined. On the other hand, of the five who died an autopsy was made in Case XL., but revealed no hydrocephalus, and the records of Cases XXIV., XXXIX., XLV., and LXXXI. are not of enough detail to warrant a diagnosis. Whether the eleven patients with symptoms of hydrocephalus, who were discharged from the hospital later, remained well is not known. We do know that Case V. of our series was discharged on the twenty-third day from the onset of his illness, yet died of hydrocephalus on the sixtieth day.

TREATMENT. Several of the probable cases of hydrocephalus just mentioned which recovered were tapped one or more times, but nothing indicates that this was of especial benefit. No large amount is reported removed and no marked amelioration of symptoms noted. The treatment of the fatal cases was purely supportive and symptomatic, and this holds good for the others. In the literature recently there have been many reports on the efficacy of lumbar puncture in hydrocephalus

[1] Cyclopædia of the Practice of Medicine, vol. ii., p. 728.
[2] Report of the State Board of Health of Massachusetts, p. 129.

after meningitis, but a study of these has failed to convince me of its usefulness. And how improvement can take place by this or any other means is very hard to see. The sudden death within twenty-four hours in Case V., following unusual exertion after a long period of convalescence, shows how careful one must be in the care even at a time when a cure appears established.

A CRITICAL SUMMARY OF THE LITERATURE

ON THE

DIAGNOSTIC AND THERAPEUTIC VALUE OF LUMBAR PUNCTURE.

By Alfred Hand, Jr., M.D.,
PATHOLOGIST AND DISPENSARY PHYSICIAN TO THE CHILDREN'S HOSPITAL, PHILADELPHIA.

Since H. Quincke's[1] epoch-making contribution in 1891, which showed appreciation of a hitherto unused anatomical fact and opened up a wide realm for investigation, there has accumulated a vast amount of literature dealing with removal of cerebro-spinal fluid through a needle introduced into the subarachnoid space between the transverse processes of the lumbar vertebræ, or, as it is briefly expressed, lumbar puncture. So extensive is the literature that only those articles will be here referred to which bring out the different points of the subject, the conclusions being based on a general review of all accessible articles.

R. Neurath[2] has given a very exhaustive review of the subject up to 1898; and while this article will, to a certain extent, be supplementary to Neurath's, it will, of necessity, touch on many of the articles which appeared prior to two years ago.

Quincke first used the operation for the relief of excessive intracranial pressure in cases of chronic hydrocephalus, to supplant the more formal operation of tapping the ventricles, which procedure necessarily often caused trauma to a limited region of brain-substance. From this as a start, the field of lumbar puncture has widened, so that it may now be said to have therapeutic and diagnostic indications. The therapeutic indications are: (*a*) to relieve excessive pressure by the cerebro-spinal fluid on the brain and cord; (*b*) to remove active deleterious bodies which may be present in the cerebro-spinal fluid.

The causes of increased tension in the cerebro-spinal fluid are: all

[1] Verhandlung. des Congresses f. innere Medicin, 1891, x.
[2] Centralblatt f. die Grenzgebieten der Medizin und Chirurgie, vol. i., 1897–98.

inflammations of the internal or external membranes, including hydrocephalus; tumor; chlorosis; traumatism; toxic states, such as those produced by lead, uræmia, or the infectious fevers. In estimating with the manometer the pressure of the cerebro-spinal fluid as it flowed through the needle, observers were at the start met with the difficulty that the normal degree of pressure in man was unknown, and when measurements were then made of it, wide variations were found to occur in health under varying conditions. Then conflicting reports were made of the pressure existing in diseased conditions, some observers reporting the finding of excessive pressure, others, in the same disease, a normal degree, while others failed to obtain fluid, which is sometimes an indication of the absence of any pressure. Order, to a large extent, has been brought out of this chaos by the valuable report of M. Pfaundler.[1] He has shown that the mistake originally was in looking upon the pressure as a unit in itself, whereas it is really made up of three elements, hydrostatic, vascular and elastic. The hydrostatic element is that pressure which the column of cerebro-spinal liquid exerts when the patient is in the sitting posture and disappears when he lies down. The element of elasticity is that furnished by the walls of the subarachnoid space and of the ventricles, which, when the amount of fluid is increased, tend by reason of their resiliency to compress the fluid; because of the bony case surrounding the brain and cord, distention of the cavities is limited, and the elastic element is therefore not great; it can be measured only during a puncture made post-mortem with the body recumbent, and usually measures about 2 mm. of mercury, although distention may increase it to 10 mm. The most important element in the pressure is the vascular, or that carried over from the heart through the bloodvessels, and it is to this that the symptoms of pressure are mainly due.

Pfaundler has estimated the normal subarachnoid pressure in the sitting posture to be from 20 to 25 mm. of mercury, about half of which is hydrostatic and disappears when the patient lies down. Estimation of the pressure by the manometer is, however, feasible only in hospital work, and it is not of so much concern to the general practitioner as the other points of the operation.

The technique is simple, and remains practically the same as suggested by Quincke, consisting of surgical cleanliness, introduction of the needle between the third and fourth lumbar vertebræ, 5 to 10 mm. to one side of the middle line, the direction of the needle being slightly toward the middle line, and in older children and in adults, slightly upward; the necessary penetration varies with the age and state of nutrition of the patient, and can be determined by the sense of touch which will tell when the cavity is reached; usually, 2 cm. in children and 4 to 6 cm. in

[1] Jahrbuch f. Kinderheilkunde, 1889, Bd. xlix.

adults are the required depths. Variations occasionally necessary from this technique are more fully described by L. A. Conner.[1]

THE DIAGNOSTIC VALUE. It is necessary at the start, for purposes of comparison, to know the characteristics of normal cerebro-spinal fluid, and thanks to the researches of A. H. Wentwoth,[2] E. Stadelmann,[3] H. Lenhartz,[4] C. Comba[5] and others it has been found to be a faintly alkaline, sterile, perfectly clear and colorless fluid, with a specific gravity ranging from 1001 to 1009, containing albumin and sugar and free from cells and other morphologic elements.

Comba has studied especially the diagnostic value of the chemical examination for sugar, with these conclusions:

1. In cerebro-spinal fluid drawn during life from children not attacked with meningitis there exists constantly a glucose-like reducing substance. The average quantity is 4 to 5 centigrammes per 100. In grave pneumonic processes it is increased a little.

2. In tuberculous meningitis the glucose is found in small amount at the start, and is absent toward the end.

3. In the meningitis due to the meningococcus of Weichselbaum and to the diplococcus of Fraenkel the absence of glucose in the exudate is constant.

4. The diminution and disappearance of glucose in the cerebro-spinal fluid are probably due more to the glycolytic action of the nucleoproteids of the leucocytes than to that of the bacteria contained in the exudate.

5. The proportion of glucose is less than that of the blood (5 to 15 cgm. per 100, according to Bunge), which tends to prove that it is a product of secretion rather than of transudation.

The amount of albumin in normal fluid is usually given as 0.2 part in 1000, the highest amount reported being 1 in 1000. Most observers agree with Lenhartz's statement that over 0.25 per 1000 speaks for inflammation, there being to this a few exceptions, as amounts of 2 to 4 per 1000 have been observed in tumor and apoplexy. While the usual amount in meningitis is about 2 to 3 per 1000, in purulent meningitis, tumors, or hemorrhage it may be increased to 7, 8, or even 9 per 1000.

With reference to the clearness of the fluid, there is a discrepancy in the different statements. Thus, Lenhartz says the appearance is of small value and the fluid may be in meningitis either purulent, seropurulent, slightly cloudy, or perfectly clear. Felsenthal[6] says practically the same, in purulent meningitis the fluid always being turbid, while in

[1] New York Medical Journal, vol. lxxi, No. 19.
[2] Boston Medical and Surgical Journal, 1896, vol cxxv.
[3] Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie, Bd. ii.
[4] Congress f. innere Medicin, 1896, xiv.
[5] Clinica Medica, 1899; Ref. in Archives de Médecine des Enfants, May, 1900.
[6] Der Kinderarzt, 1897, viii.; Ref. in Pediatrics, vol. v., No. 3.

tuberculous meningitis it is always colorless. On the other hand, Wentworth, whose work has been very thorough, asserts positively that the fluid in meningitis is always turbid and never clear, although in tuberculous meningitis it may be necessary to compare the fluid with distilled water to notice the opalescence. The writer's rather limited experience bears out Wentworth's statement.

Fibrin, which does not form in normal fluid after it is drawn, is, according to Lichtheim[1] and Wentworth, always formed in meningitic fluid on standing. Felsenthal calls attention to this fact and to the comparison with fluid from cases of brain-tumor, in which fluid no fibrin forms. Further differential points, which may be here referred to, are that in tumor, sugar and albumin are present in normal amount, while in meningitis sugar is absent and albumin is increased. However, Freyhan[2] and Lenhartz could find no sugar in the fluid from tumor cases.

Blood in the fluid is of varied significance. Its most frequent source is a puncture of a bloodvessel by the needle, and this most commonly happens by the needle being pushed too far, in which case the venous plexus which lies behind the bodies of the vertebræ is injured. If the blood is in the first part of the liquid, the remainder being clear, traumatism by the needle is probably the source, especially if, as pointed out by Fuerbringer,[3] immediate microscopic examination shows normal, unchanged erythrocytes. Other causes for an admixture of blood in the fluid are injuries to the head or spine, apoplexies, subdural or rupturing into the ventricles; occasionally meningitis (Councilman[4]).

The cellular elements in the fluid are, according to Wentworth, the cause of the turbidity; in tuberculous meningitis they are mainly small, round, mononuclear leucocytes; in purulent meningitis the polynuclear cells are in the majority. Endothelial cells are also present sometimes.

Of greater importance than the physical or chemical examination of cerebro-spinal fluid are the positive findings of bacteria; the importance of negative results in the search for them depends largely on the technique and skill of the examiner, so that, as a rule, the statement put forth by Stadelmann holds good, that positive conclusions can only be drawn from positive and never from negative results.

The following bacteria have been found in cerebro-spinal fluid:[5]

1. Staphylococci. Josias and Netter[6] found the staphylococcus pyogenes aureus in a case of general infection, as did Wentworth.

2. Streptococci.

[1] Berliner klinische Wochenschrift, 1895.
[2] Ibid.
[3] Congress f. innere Medicin, xv.
[4] Transactions of the Association of American Physicians, 1897.
[5] For the names of the observers and references up to 1898, the reader is referred to Neurath's review.
[6] Gazette des Maladies Infantiles, 1899, No. 23; Ref. in Archives of Pediatrics, January, 1900.

3. Pneumococci. The Fraenkel-Weichselbaum diplococcus lanceolatus.

4. Meningococci. The Weichselbaum-Jaeger diplococcus intracellularis meningitidis. Of great interest is the finding by F. Schultze[1] of this germ in pure culture from the cerebro-spinal fluid of a five-year-old boy attacked with acute anterior poliomyelitis.

5. Typhoid bacilli. In all the literature but two findings of this germ are reported, the first by Lenhartz[2] and the other by Wentworth,[3] who observed a case of typhoid fever in a girl four years old, the Widal test being negative on the seventeenth and twenty-first days; on the twenty-second day signs of meningitis developed, followed by death in fourteen hours. There was no autopsy, but a post-mortem lumbar puncture gave a turbid liquid containing many bacilli decolorized by Gram; cultures of this germ gave agglutination with serum from two cases of typhoid fever.

6. Bacterium coli.

7. Tubercle bacilli. With regard to the examination for this germ the extremes of opinion are represented by Monti[4] on the one hand and Wentworth on the other. The former states that lumbar puncture furnishes no help in the diagnosis of tuberculous meningitis, and reports fifteen cases, in the fluid from none of which were tubercle bacilli found. Wentworth states that a general review of the literature convinces him that the technique is often at fault, and that with skill in examination, if this examination includes staining culture, and inoculation, positive results in meningitic fluid will almost never be missed. In this connection it is interesting to note (and the writer's own experience bears this out) that the cases of tuberculous meningitis in which the tubercle bacilli were not found were among the earliest examined, and it is safe to infer that as the skill of the examiner increases positive findings become more frequent. Thus, Lenhartz found the germs in the last nine of nineteen cases. His method is to let a bit of absorbent cotton sink to the bottom of the fluid, fish it out and tap it on the cover-glass, then staining for tubercle bacilli. The writer has found to be equally satisfactory the method recommended by Fuerbringer, which is to allow the fluid to stand for some hours until the slight net of fibrin which forms has sunk to the bottom; this is picked out with a platinum needle fixed on glass and stained. The method freest from exceptions, however, as urged by many writers, is the injection of the fluid into a guinea-pig, the objection being the time which must necessarily elapse, four to six weeks, before the results can be obtained.

The positive finding, by staining, of other bacteria than the tubercle

[1] Münchener medicinische Wochenschrift, September 20, 1898.
[2] Congress f. innere Medicin, 1897, xv.
[3] Archives of Pediatrics, November, 1899.
[4] Archiv. f. Kinderheilkunde, Bd. xxiv.

bacillus is open to the objection that these bacteria may not have the power of growth, and Stadelmann therefore urges that cultures and inoculations should always be made before positive conclusions are drawn.

The inability to draw positive conclusions from negative findings lessens the value of lumbar puncture for guidance in cases of brain abscess; evidences in the fluid of meningitis would, of course, contraindicate operation, but a negative result does not always exclude meningitis, for the reason that inflammation may have occluded the foramen of Majendie or the subarachnoid space at the base of the brain, and ependymitis or meningitis might exist without any evidence of the spinal fluid.

As a diagnostic means it remains to speak of the findings of Pfaundler concerning the pressure of the fluid. Space does not permit mention of all his results, but of importance is the characteristic curve which he describes for tuberculous meningitis, as follows: In the stage of brain-irritation, 48 mm. of mercury; in the stage of brain-pressure, 52 mm.; in the stage of brain-paralysis, 24 mm.; immediately after death, 11 mm.

The Therapeutics of Lumbar Puncture. As a therapeutic means lumbar puncture may be either palliative or curative. When the pressure of the fluid, from any cause whatever, is so great as to threaten life, the lowering of this by lumbar puncture is a palliative procedure which may even be called temporarily curative, even in incurable conditions. This is often seen in tuberculous meningitis, the operation being followed by a comparative degree of comfort, with lessening of the headache, the cries and contractures. The operation has not settled yet the question as to whether recovery ever follows tuberculous meningitis, the case reported by Freyhan[1] still lacking confirmation, unless we except Fuerbringer's patient, who recovered, and in whose cerebro-spinal fluid tubercle bacilli were found, the diagnosis of the actual existence of meningitis in these cases not being absolutely established.

The influence of lumbar puncture on the course of epidemic cerebro-spinal meningitis is held almost unanimously by all observers to be beneficial, if practised early and repeated often. The case reported by Netter[2] will serve as an illustration: From the second to the thirty-seventh day eleven lumbar punctures were made at intervals of two to five days, 30 to 70 c.c. being drawn at each time. The first puncture gave a turbid fluid with a deposit of pus; the second, two days later, fluid with a slight sediment; and the third, fluid with a very small fibrinous flake. After this the liquid became perfectly clear, and the author is of the opinion that the withdrawal of pus probably was of benefit.

[1] Deutsche medicinische Wochenschrift, 1894.
[2] Archives de Médecine des Enfants, July, 1900.

Few observers have seen any good follow the operation in cases of brain-tumor, occasionally there being temporary lessening of the headaches and improvement in the choked disk. It is only in tumor, and that, too, only in adults that lumbar puncture has been followed by untoward results, a number of cases of sudden deaths having been reported. Noelke[1] seeks to explain these, in all of which the tumor had been situated at or near the base, by showing that the withdrawal of the fluid from the spinal canal allows the tumor to sink like a stopper toward the foramen magnum and to press on the vital centres.

The most that can be said for lumbar puncture in chronic hydrocephalus is that it accomplishes as much as ventricular aspiration, the value of either being doubtful.

Fear of favoring hemorrhage by lowering the pressure of the cerebro-spinal fluid too much, and absence of beneficial results have led to the abandonment of the operation in apoplexy, hemorrhagic pachymeningitis, softening, and embolism.

Reports of the operation in uræmia are not numerous, and the beneficial results in some cases could not be positively ascribed to the puncture.

Seegelken[2] has reported a case of saturnine encephalopathy in which the coma and unilateral convulsions disappeared after lumbar puncture, and recovery followed.

In chlorosis the severe headaches have been relieved, according to many reports, by the withdrawal of as much as 70 c.c. of fluid.

After finding increased pressure in cases of infectious diseases, in which there were head-symptoms, Lenhartz, Stadelmann and others recommend the use of lumbar puncture after other means of relief have been unsuccessful.

In two cases of traumatism to the spine, followed by paraplegia, G. W. Jacoby[3] removed by puncture bloody fluid, and rapid improvement followed.

With the above facts before us, it seems safe at present to draw the following conclusions:

1. Lumbar puncture has a wider field as a diagnostic aid than as a therapeutic means.

2. As an aid to diagnosis lumbar puncture is of value only when examination of the fluid gives positive results; it is not safe to draw conclusions from negative results.

3. Therapeutically it is of value in epidemic cerebro-spinal meningitis to bring about recovery; in tuberculous meningitis to promote comfort; and in other conditions of excessive pressure to favor recovery by removing a condition immediately dangerous to life.

[1] Deutsche medicinische Wochenschrift, 1897.
[2] Münchener medicinische Wochenschrift, 1896.
[3] New York Medical Journal, vol. lxii., 1895.

REVIEWS.

THE TREATMENT OF FRACTURES. By CHARLES LOCKE SCUDDER, M.D., Surgeon to the Massachusetts General Hospital, Out-patient Department; Assistant in Clinical and Operative Surgery in the Harvard Medical School. Assisted by FREDERICK J. COTTON, M.D. With 585 illustrations. Philadelphia: W. B. Saunders & Co.

FRACTURES. By CARL BECK, M.D., Visiting Surgeon to St. Mark's Hospital and to the New York German Poliklinic; formerly Professor of Surgery, New York School of Clinical Medicine, etc. With an Appendix on the Practical Use of the Röntgen Rays. With 178 illustrations. Philadelphia: W. B. Saunders & Co., 1900.

IT is with a feeling of relief that we welcome two new books that treat of non-operative surgery. Operative surgery has occupied so much of the attention of the profession during the past fifteen or more years that the non-operative work has suffered from neglect. The development of antiseptic technique has so broadened the sphere of operative work that it has seriously encroached not only on the heretofore non-operative surgical affections, such as fractures, dislocations, hernia, etc., but even on what were regarded as medical affections proper—inflammatory affections of the region of the cæcum, typhoid fever, diseases of the kidney, and others. This operative advance has invaded the treatment of fractures, but to a limited extent only, and the bulk of cases still are treated by conservative measures. It is now four years since the announcement of the discovery of the X-ray by Prof. Röntgen, and its use has been sufficiently prevalent to cause the issue of some works on fractures which deal largely or almost entirely on this aspect of the subject. A dispassionate review of the results of the employment of the X-rays in fractures will convince one that our earlier expectations were too sanguine. While the information obtained by this method of examination is always of interest, and often of service both in diagnosis and treatment, in comparatively few cases is it absolutely essential. Inasmuch as X-ray examinations are expensive and only obtainable, as a rule, by those who have access to hospitals, it is evident that the bulk of fractures seen by private practitioners, particularly those located in the country districts, will have to be treated without the aid which they afford. On this account it is in the highest degree inadvisable to place too great reliance on them and neglect the other and more accessible means of diagnosis. It seems to me that there has been a lessening in the ability of our young men particularly in diagnosing the lesions in fractures. The internes of our hospitals, instead of establishing their diagnosis by a careful physical examination of the injured part, are too apt to defer making a diagnosis on the plea of not wishing to distress the patient, and rely on the X-ray examination.

In both the works before us skiagraphs occupy prominent positions. The work of Scudder and Cotton produces a favorable impression by the general manner in which the subject is treated. The volume is not intended apparently as an elaborate treatise, but rather as a handbook to which one can refer to obtain a reliable and modern method of treatment, and as such it is satisfactory. Its descriptions are concise and clear and the treatment sound. The physical examination of the injured part is well described, and in several cases, as in injuries of the elbow, wrist, and other parts, the method of making these examinations is illustrated by a liberal use of cuts. This makes the book particularly valuable to those who have not access to X-ray outfits. We believe the authors are right when they advocate in general the use of splints as a dressing in preference to plaster-of-Paris. The use of the latter in suitable cases is often alluded to and properly explained, but not to the exclusion of splints. It is certainly apt to prove unreliable in the hands of those not well accustomed to its application. The authors are not enthusiastic over the ambulatory method of treatment, and we again believe them to be right. The use of the Taylor hip splint is said to be of distinct value in fractures of the femur. The treatment may also be adopted in certain carefully selected cases of fractures below the knee, particularly of the fibula. The resort to operative measures is recommended in suitable cases. While not especially radical, the advice is sound. They advise in fracture of the patella that operation should be confined to healthy individuals under sixty years of age; to fractures with a separation of an inch or more of the bony fragments and extensive lateral fascial tears, and to cases presenting great distention that does not disappear quickly.

The question of the value of massage is hardly sufficiently insisted on. While the treatment of fractures solely by massage, as has been advocated abroad, is not to be advised, it is undoubtedly true that massage is not employed in this country in the treatment of fractures to anything like the extent it should be. Fractures are allowed to remain in splints or plaster for weeks without any attempt being made to prevent stiffness by the use of massage.

There is a separate chapter by E. A. Codman on the Röntgen Ray and its Relation to Fractures. In it particular attention is drawn to the necessity of avoiding mistakes in interpreting skiagraphs.

It is interesting to note that the authors advise the treatment of fractures of the elbow in the fully flexed position.

The book as a whole is mainly a reflection of the views of the authors and of the practice prevalent in their neighborhood. The work of others is comparatively seldom alluded to. As far as it goes the work is a good one; we like it, and think others will; but it is in a different class from the classic treatises of Stimson and Hamilton.

The work by Carl Beck is smaller than that of Scudder's, and still more narrow in its treatment of the subject. He dedicates his book "To Wilhelm Conrad Röntgen, without whose discovery much of this book could not have been written." He thanks Professor Röntgen for the many kindnesses extended to him at his laboratory, so that it is no surprise to find that great attention is paid to X-ray illustrations. The book is written essentially from the stand-point of one who relies largely on the uses of the X-rays in practice.

He defines a fracture as a solution in the continuity of a bone, and divides fractures into spontaneous and traumatic. This suggests the question whether it is not more correct to say that a fracture is a solution of continuity due to violence? A simple solution, such as occurs in caries, is no fracture, and in spontaneous fractures the breaking is still the result of violence; therefore, they, too, are traumatic fractures. From the fact that the author has already written a book on surgical asepsis, we might expect that his views as to operative procedures would be radical; but such is not the case. He says: "There has lately been observable a tendency on the part of a few surgeons to treat simple subcutaneous fractures by wiring the fragments. While under the auspices of asepsis such treatment need not be followed by any reaction, and might in the hands of competent masters give excellent results in suitable cases, such tendencies must be regarded as surgical aberrations. It is only where much diastasis is present, as in fractures of the patella (olecranon), when bony union appears improbable, that such vigorous interference is demanded. But by our recent means of making a positive diagnosis possible in all cases it is usually just as easy to obtain a perfect result by simple bloodless reduction and by thorough immobilization."

He states that massage is a splendid adjunct in the after-treatment of fractures, but to substitute it entirely for the good old immobilization treatment, as has been advocated recently, is not advisable. After mentioning some of the advantages of the ambulatory dressing, he proceeds as follows: "But these advantages are fairly offset by the immense difficulty in keeping the treatment under permanent control in practice. The technique of applying such dressings is complicated, and, therefore, dangerous in the hands of the inexperienced. In hospital practice, where continuous control is possible, the adoption of this method in many instances proves to be of great value. So, while this treatment is undoubtedly advisable in cases in which the dressing can be removed at any moment, in case ischæmic symptoms should manifest themselves, it should not be recommended for adoption in general practice."

In speaking of fractures of the condyles of the humerus he alludes to them as fractures of the epicondyles. What American anatomists and surgeons call condyles the Germans call epicondyles. To avoid this confusion of terms the reviewer, in the *Annals of Surgery*, January, 1899, suggested that one speak of intracapsular and extracapsular fractures of the condyles, and drop the name epicondyle entirely. Our author, however, speaks of intracapsular epicondylar fracture and isolated epicondylar fracture. In regard to treatment of fractures in the region of the elbow, he places them in almost any position and applies usually plaster-of-Paris, or sometimes wooden splints, and relies on frequent examinations by the X-rays to assure him that the fragments are in proper position. The utter lack of exactness and detail renders his descriptions of treatment of little value except to one who has already had an extensive experience. The same feature exists all through the book and impairs its usefulness for the general practitioner very much.

To sum up, the book will be valuable to those who wish to be informed as to the use of the Röntgen rays in the treatment of fractures, but as a guide for the inexperienced, either for treatment or to obtain

a general knowledge of the subject, it will be found too superficial and limited in both its descriptions and directions.

Both volumes are published in excellent style, on thick, highly calendered paper that brings out the illustrations well. G. G. D.

A MANUAL OF OBSTETRICS. By A. F. A. KING, A.M., M.D., Professor of Obstetrics and Diseases of Women and Children in the Medical Department of the Columbian University, Washington, D.C., and in the University of Vermont; Obstetrician to the Columbian University Hospital. Philadelphia and New York: Lea Brothers & Co., 1900.

A MOST excellent book. It could hardly fail to be and still reach its eighth edition. The clearness of expression and elimination of unimportant details which made the earlier editions so welcome to the student are still evident in the present volume.

The whole volume is good, but if it were necessary to pick out any particular chapters as better than the rest, those on Puerperal Sepsis, Mechanism of Labor, and the Jurisprudence of Midwifery would occur to one.

In the case of such a book, thoroughly satisfactory as it is for the most part, it seems almost a pity to criticise, and yet there are certain statements which cannot be overlooked. For instance, in speaking of the treatment of the pernicious vomiting of pregnancy, the statement is made that a woman can be nourished for weeks by rectal alimentation alone. This certainly is dangerous teaching, as it might readily lead the inexperienced practitioner to put too much trust in the rectal tube, and thus defer the induction of abortion until too late.

Again, the weight of testimony is certainly against the use of the vaginal douche after the completion of a normal case of labor. Exception must also be taken to the method advised by the author for the purpose of rendering the vagina sterile, experience having certainly shown that the use of a bichloride douche alone is not at all as certain as when it is preceded by a thorough cleansing with soap and water.

It seems unfortunate that more space was not given to the artificial feeding of infants, there being probably no more troublesome duty devolving upon the physician in the course of his obstetrical work. The space used in the description of the operation of laparo-elytrotomy might well have been so utilized.

One of the most unfortunate statements of the book is that it is impossible to reach the promontory in the normal pelvis. This, certainly a mistake, might well involve the student in serious difficulty. In this same connection the advice given to insert the whole hand in the vagina in order to measure the true conjugate can only be condemned.

The description of the operation of version and the indications for its performance is very good, but it is hard to understand why it is advised to grasp and bring down both feet if possible, as by so doing a much less efficient dilatation of the soft structures is secured.

It is also to be deplored that the author gave prominence to the preparations of iron in the treatment of post-partal bleeding, and it is to be hoped that no one will practice obstetrics without realizing that mouth-to-mouth insufflation is at least as good a method of resuscitation of

asphyxiated children as is Sylvester's. Why the author teaches that Cæsarean section is the treatment to be advised as a means of delivery at the moment of maternal death or shortly before is difficult to understand, as experience proves that version gives in such emergencies much better chances for saving the infant's life, and is particularly easy of performance, as the parts are relaxed by the approach of death.

The treatment of the lacerations of the perineum does not receive the attention which the unfortunate frequency of this injury would seem to warrant. It is true that a method of repair of median tears, both with and without involvement of the rectum, is given; but the student could hardly hope to treat this condition successfully if he had no more complete knowledge than is contained in the description of the author, since there is no method given for repair of the tears of the sulci, although they are more serious than the median tears, and much more likely to be met with than either the so-called second-degree tear of the external perineum or the tear involving the rectum. W. R. N.

HAND-BOOK OF OPTICS, FOR STUDENTS OF OPHTHALMOLOGY. By WILLIAM NORWOOD SUTER, B.A., M D. New York: The MacMillan Company. London: MacMillan & Co., Ltd., 1899.

THE pages of manuals upon the eye which deal purely with optics are, as a rule, scanned but cursorily by the average student of ophthalmology, as he shuns the study of this branch of his art as a thing full of symbols and signs, quite unintelligible to his intellect, and, as he thinks, without any particular value in actual practice. The reason for this indifference probably lies in the fact that hitherto it was possible to approach these fields only through the higher mathematics—channels too deep for the average brain. In the excellent little work before us Dr. Suter has solved these problems by means of simple algebra and elementary geometry alone, and has made the entire subject of optics understandable even to the student who approaches the study of ophthalmology with but little previous mathematical training. The book contains no more than every practising ophthalmologist should be familiar with, and we urge its careful study by all. W. C. P.

REFRACTION AND HOW TO REFRACT, INCLUDING SECTIONS ON OPTICS, RETINOSCOPY, THE FITTING OF SPECTACLES AND EYE-GLASSES, ETC. By JAMES THORINGTON, A.M , M.D. With 200 illustrations, 13 of which are colored. Philadelphia: P. Blakiston's Son & Co., 1900.

DR. THORINGTON has the happy faculty of appealing directly to the student's mind when he writes, and the book before us will undoubtedly meet with the same favor as his earlier works on retinoscopy. Unlike the book which has just been reviewed, the author touches but lightly upon the problems of physiological optics, merely giving sufficient principles to make his remarks upon the refraction of the eye understandable.

His advice in regard to the treatment of anomalies in the extra-ocular muscle balance is conservative and sound; especially would we commend his caution in regard to the performance of tenotomies.

W. C. P.

THE REFRACTION OF THE EYE, INCLUDING A COMPLETE TREATISE ON OPHTHALMOMETRY. A CLINICAL TEXT-BOOK FOR STUDENTS AND PRACTITIONERS. By A. EDWARD DAVIS, A.M., M.D. With 119 engravings, 97 of which are original. New York: The MacMillan Company. London: MacMillan & Co., Ltd., 1900.

THE reviewer regrets that he is unable to extend the same words of praise to this work as he has to the two preceding. He views any work which contains in its preface the statement "that incidentally throughout the book I have endeavored to show the utter uselessness of a mydriatic in fitting glasses in the vast majority of cases, even in young subjects," a very dangerous guide to students of ophthalmology. Had the author entitled his book "ophthalmology," and had he limited himself to the exposition of the ophthalmometer, as it is with this phase of the subject of refraction that he chiefly deals, the work might, perhaps, have been acceptable. The author makes a point of inserting the histories of a number of illustrative cases to elucidate practical points in the use of the ophthalmometer, but, unfortunately, the majority of them are rendered worthless for scientific purposes by the meagreness of their data. The book has been published in a most attractive manner, which is unfortunate, as its appearance would lead the tyro to estimate it as a work of more importance than its text fails to substantiate.

W. C. P.

A MANUAL OF THE DIAGNOSIS AND TREATMENT OF THE DISEASES OF THE EYE. By EDWARD JACKSON, A.M., M.D. With 178 illustrations and 2 colored plates. Philadelphia: W. B. Saunders, 1900.

THE science of ophthalmology has been well expounded during the past decade by a series of remarkable text-books, so that there scarcely seems to be any need at present for an additional work upon the subject, unless every phase of it was treated in an original manner. The reviewer confesses to a degree of disappointment, therefore, in the book before him. Dr. Jackson is one of the best known of American ophthalmologists, and as his work heretofore has been characterized by originality, the profession has been led to expect that his text-book would bear this stamp, and that it would differ somewhat from other books of a similar character. The work, however, imitates its predecessors, and while the easy style in which the manual is written, and the explanations of the text, especially of that portion which deals with the refraction of the eye, make the work particularly acceptable to beginners in ophthalmology, it is to be regretted that the author did not embrace the opportunity of attempting a more comprehensive treatise.

W. C. P.

A TEXT-BOOK OF THE DISEASES OF WOMEN. By CHARLES B. PENROSE, M.D., Ph.D., Professor of Gynecology in the University of Pennsylvania; Surgeon to the Gynecean Hospital, Philadelphia. Illustrated. Third edition, revised. Pp. 531. Philadelphia: W. B. Saunders & Co., 1900.

THE appearance of three editions of a text-book on gynecology within less than three years is a sufficient proof of its inherent excellence. Although we have already called attention to its peculiar value as a practical book for the student, we feel that the new edition should not be dismissed without further commendation.

Viewed from a teacher's stand-point, its most pleasing feature is the absence of any attempt on the part of the author to intrude his own fads. Keeping steadily in view the purpose so modestly expressed in the preface, "to avoid confusing the student" by the introduction of anatomical and pathological details, he has presented each subject so concisely and yet so lucidly that the beginner cannot fail to grasp the essential facts.

It has often been said that there is no call for new text-books on diseases of women, because they must necessarily be mere compilations from the old. But, while in one sense this is true, there is always room for one in which old matter is presented in a new and attractive form. The student has no time for theories and disputed points in practice. What he needs is a few facts which will *stick*. This point the author thoroughly understands, and he never loses sight of it.

The chapters on lacerations of the perineum and pelvic floor are models of accurate description and sound teaching, while those on uterine displacements admit of no criticism. Laceration of the cervix uteri, a subject the importance of which has been underestimated by foreign gynecologists, is admirably handled. The critical reader will be less pleased with the succeeding chapter on cervical catarrh. The present generation of medical students should certainly be grateful to the author for omitting the usual obscure discussion of the condition variously described as "chronic metritis," "areolar hyperplasia," etc. He wisely evades this issue in a sentence on page 200, viz.: "The pathological changes that take place resemble those occurring in chronic inflammation in similar musculo fibrous structures in other parts of the body." Uterine neoplasms are described in an especially pleasing way, and the subject of diseases of the tubes is presented in the most approved manner. We note with satisfaction the author's attitude toward conservative operations. His statement that "successful cases show the possibilities of surgery, but, unfortunately, they are exceptional," will meet with the approval of those who are not entirely carried away with enthusiasm for the prevailing fad. As an illustration of the writer's faculty of elucidating a difficult subject in a few well-chosen sentences we refer the reader to the section on pelvic suppuration on page 296. Other topics which are skilfully handled are diseases of the ovaries, diseases of the urethra and bladder, and gonorrhœa. The concluding chapters on operative technique and after-treatment are full of practical suggestions. We are surprised to note that the use of the ancient glass drainage-tube is still favored.

In conclusion we can only reaffirm our belief that Dr. Penrose, without making any pretensions to writing an extended work on gynecology, has rendered a distinct service to his generation by giving us what approaches very closely to the ideal text-book. We predict that its superiority will be widely recognized by teachers as well as students. H. C. C.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

WILLIAM OSLER, M.D.,

PROFESSOR OF MEDICINE IN THE JOHNS HOPKINS UNIVERSITY, BALTIMORE, MARYLAND,

AND

GEORGE DOCK, M.D.,

PROFESSOR OF MEDICINE IN THE UNIVERSITY OF MICHIGAN.

Arteritis in Relation to Enteric Fever.—AUDEN (*St. Bartholomew's Hospital Reports*, 1898, vol. xxxv., p. 55) draws attention to the very rare occurrence of gangrene as a complication of typhoid fever. This is borne out by the fact that neither Trousseau nor Flint saw a case, and Murchison, in his long experience at the London Fever Hospital, met with only one instance.

Auden reports three instances in which this complication developed in typhoid patients treated at St. Bartholomew's Hospital during a period of eighteen months. In Case I. there was a thrombosis of the right femoral artery, followed by gangrene, and requiring amputation of the leg at the junction of the upper and middle thirds. In Case II. there was bilateral thrombosis of the popliteal arteries, causing gangrene and necessitating the amputation of both feet. Case III. was not one of actual gangrene. There was definite thrombosis of the left femoral artery, with cyanosis of the skin and the other usual accompanying symptoms, but without actual gangrene.

The writer has collected and tabulated eighteen other cases, making a total of twenty-one. Ferrand had collected twenty-three cases, but as six cases occur in both groups, it would make a total of thirty-eight cases. Auden's study is based on an analysis of these cases.

The most striking feature brought out is the frequency of the complication in early adult life. The oldest patient in the series was thirty-four, and only three had reached the age of thirty. It is to be remembered that a very large percentage of typhoid occurs between fifteen and thirty years of age.

The complication is practically twice as frequent in males as in females. Of the 38 cases no less than 26 were in males. This may in part be due to the greater frequency of typhoid fever in males than in females. From 1887 to

1897 the proportion of males to females with typhoid at St. Bartholomew's was 59.9 to 40.1.

The affection is almost always confined to the lower limbs. In only 3 of Auden's 21 cases were arteries of the upper extremity involved. The right leg is more frequently affected than the left. The complication is essentially one of convalescence, the average day of appearance being the thirtieth day. The mortality cannot be considered high for so severe a complication. Of the 38 cases death occurred in only 13, *i. e.*, there were 65.8 per cent. of recoveries.

Concerning the origin of the gangrene there are two main theories. The shutting off of the blood supply is, according to Auden, due either to a primary arteritis, with the subsequent formation of a clot, or to the lodgement of an embolus, with the evidence in favor of the former. He admits that embolism is probably the cause in some cases, because acute endocarditis does occur in typhoid fever. It was present in 11 of the 2000 Munich autopsies. The tendency to dilatation of the heart in typhoid, with the consequent predisposition to clot formation, would favor the chances of the gangrene being due to embolism.

As already stated, Auden favors the primary arteritis theory as being the probable cause of the clotting and gangrene in most cases. Unfortunately he is not able to bring very substantial proof to support his belief. He points to the diffuse proliferation of endothelial cells in the minute capillaries of the intestinal walls, which eventually causes obliteration of the vessels and consequent necrosis of the Peyer's patches. He further refers to the observation of Mallory, who states that frequently during the regressive period, in the collection of cells beneath the epithelium, necrosis of large phagocytic cells takes place, and furnishes the starting-point for a very abundant formation of fibrin, which extends for some distance along the vessel, even reaching out into the lumen and dividing it into two or more channels. The cells of the blood current become entangled in this fibrin, and lead to the rapid formation of thrombi, which completely occlude the vessel. Auden thinks that this observation in connection with the intestinal vessels is of great importance, and believes that a somewhat analogous process occurs in the peripheral arteries. He believes that the path by which the arterial wall is infected is through the vasa vasorum, into which the bacteria pass. He admits that the finding of typhoid bacilli in the thrombus does not determine its embolic or autochthonous character, for it is certain that a clot forming as an infected area of arterial wall will itself become secondarily infected. Unfortunately there is no report of a bacteriological examination in any of the published cases.

The possibility of the thrombus being of the nature of a marantic thrombus or being due to blood changes is entertained.

Report on the Cases of Typhoid Fever Admitted into the Royal Victoria Hospital, Montreal, During the Year 1899.—GILLIES (*The Montreal Medical Journal*, June, 1900, p. 422) has analyzed the typhoid fever cases admitted to the Royal Victoria Hospital, Montreal, during 1899. In all, there were ninety eight cases admitted. Of these, eighty-six were treated to a conclusion, and the percentages given in the report are based on this

number. Of the remaining twelve cases several were cured, and the others were convalescent, and it is to be inferred that they still remained in the Hospital at the end of 1899.

The largest number of cases was admitted during the month of August, the smallest during December.

A few of the percentages given may here be referred to. The shortest, average, and longest duration of fever were eight, twenty-four, and fifty-eight days respectively.

The onset of symptoms was gradual in 95.4 per cent. of cases. Chills occurred at the time of onset or during the first week in 17.4 per cent. Diarrhœa was present at the onset in 20.9 per cent., and continued throughout in 8 per cent. Epistaxis and vomiting occurred at the onset in 18.6 and 34 per cent. of the cases respectively. Delirium occurred at the onset or during the course of the fever in 18.6 per cent of the cases.

An eruption was present in 72 per cent. of the cases. In one case the eruption was petechial; in all the rest it was of the nature of rose-spots.

The spleen was palpable in 67.4 per cent of the cases. A definite relapse occurred in eleven cases, or 12.7 per cent. The highest recorded temperature was 106.4°.

Acute bronchitis was present in 14.9 per cent.; periostitis developed during the attack in 1 case and in convalescence in another. Acute nephritis occurred in four cases. Cholecystitis occurred in three cases. Perforation of the bowel occurred twice, and intestinal hemorrhage in nine cases.

The Widal test was positive in all but four cases. In these it was negative throughout. Of these, three were regarded as abortive typhoid. No rose-spots were present. The reaction was present on the average on the eighth day. In one case it was not present till the eighteenth day.

Of the eighty-six cases, seven, or 8.16 per cent., died. Death resulted in three cases from hemorrhage, in one case from hemorrhage and perforation, in one case from perforation, and in two cases from profound intoxication.

The success in the treatment of typhoid fever at the Royal Victoria Hospital during the past six years has been very satisfactory, judging from the percentages of mortality. For the years 1894 to 1899 inclusive, the mortality was 3.5, 4.7, 0.0, 9.3, 4.3, and 8.16 respectively. During the six years 494 cases were treated, with 25 deaths, giving the low average mortality of 5 per cent.

A Report of Twenty eight Cases of Suppurative Hepatitis. During the past twenty years twenty-eight cases of hepatic abscess were admitted to the Presbyterian Hospital, New York. Owing to the small number of sailors admitted for treatment these cases are believed to represent fairly well the local conditions giving rise to the disease.

STUART HART (*Presbyterian Hospital Reports*, 1900, vol. iv., p. 150), who reports the series, groups the cases as follows according to the path by which the infection has reached the liver: (1) Entrance by way of the portal vein, seventeen cases; (2) entrance by way of the hepatic artery, three cases; (3) entrance by way of the bile passages, three cases; (4) unclassified, six cases.

Of the seventeen cases in the first group, anatomical lesions of the alimen-

tary tract were found in ten cases at autopsy, and in the other seven the symptoms gave evidence of intestinal lesions. A point of interest is the fact that in not a single case were amœbæ coli found in the stools or hepatic abscess. In six cases the abscess followed ulceration of the colon or caput coli; three cases arose from ulcerative appendicitis; one followed ulcer of the stomach. One case was believed to be of typhoid origin, owing to the blood having given the Widal reaction and the typhoid organism having been grown from the contents of the gall-bladder.

In the second group the abscesses were a part of a general pyæmic process, two of them being secondary to malignant endocarditis and the third following puerperal sepsis. From other existing pathological processes there was a possibility of infection by way of the portal vein in these cases.

In the third group the common duct was occluded in the two cases by a calculus, which was regarded as the causative agent in both instances.

Practically all the twenty-eight cases had fever of an irregular type. Chills were usually present. Icterus was present in only six cases. Pain was absent in only three cases. Tenderness over the hepatic region was usually present. The enlargement of the liver was usually upward.

In eight of the twenty-eight cases the patients were moribund on admission, and death occurred in a few hours. Of the remaining twenty only three recovered: two after evacuation of the pus by operative procedure and one by rupture into the lung and expectoration of the contents of the abscess. In all fourteen cases were operated on, with two recoveries as stated.

Autopsies were obtained in twenty of the twenty-five fatal cases. In thirteen of these there was a single abscess, making, with the three cases which recovered and in which the abscess was considered single, a total of sixteen cases in which the abscess was single. The writer states that ten of the remaining twelve cases had multiple abscesses, but it is difficult to see how this was positively determined, owing to autopsies not having been obtained in all cases. In eighteen cases the suppurative process was situated in the right lobe, more frequently in the upper part.

Concerning the So-called Pericarditic Pseudocirrhosis of the Liver.—EISENMENGER (*Weiner klinische Wochenschrift*, 1900, No. 11) has made a critical study of this condition, from which the following conclusions may be cited: It is true that severe ascites without œdema of the legs frequently occurs in consequence of an adhesive or fibrous pericarditis. If the pericarditis is latent, the symptom-complex has a certain degree of resemblance to that of cirrhosis of the liver. The symptom-complex, however, is not produced, as F. Pick claims, by connective-tissue growth in the liver due to circulatory disturbance, but is due to different causes in different cases. Important are such things as twists, compressions, or kinks of the inferior vena cava by an exudate or by fibrous adhesions of the pericardium and mediastinal tissue, and coexisting peritonitis about the portal fissure; finally, the fact that the symptom-complex occurs especially in young individuals with normal capillaries and small vessels in the greater circulation, so that œdematous transudations do not easily occur. The symptom-complex described by Pick has no simple anatomical basis, and should, therefore, not be

looked on clinically as a single disease. In addition to this, the name proposed by Pick contains a diagnostic error, and should not be perpetuated.

SURGERY.

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL;

ASSISTED BY

ALFRED C. WOOD, M.D., INSTRUCTOR IN CLINICAL SURGERY, UNIVERSITY OF PENNSYLVANIA; ASSISTANT SURGEON, UNIVERSITY HOSPITAL.

AND

C. L. LEONARD, M.D., ASSISTANT INSTRUCTOR IN CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA.

Fractures of the Neck of the Radius.—MOUCHET (*Revue de Chirurgie*, May 10, 1900) points out the fact that these fractures have been shown by the Röntgen method of diagnosis to be much more frequent than has been supposed. They are particularly liable to occur in children between the ages of nine and twelve years. His observations are based upon a series of eleven cases which have come under his observation within a few years. They are not as frequently associated with other fractures about the elbow as has been supposed, but four of these fractures were associated with other fractures. They may be complete or incomplete, though the latter are rare. The line of fracture was through the neck of the radius or just below it, and may be transverse or oblique. The ligaments are frequently ruptured. The muscles are more or less ruptured, and there is always a swelling, which, however, is frequently due to extravasations between the muscles. The amount of displacement is variable, sometimes it is absent, sometimes there is impaction, usually the lower fragment is carried anteriorly and upward by the action of the biceps muscle, but there is generally a portion of the fractured surface in contact, though usually it is only slight.

The mechanism of the production of these fractures is generally that of indirect violence, and seldom, if ever, are they due to direct force.

The attitude of the patient is almost pathognomonic. The elbow is flexed at an obtuse angle or may be at a right angle. The hand is in pronation or semi-pronation and supported by the other hand. It is never in complete supination. There is a swelling of the forearm muscles, due to exudates and not to the displacement. It varies in amount. Ecchymosis is a late sign. Pain is probably the most constant and reliable symptom. Its position and the method of elicitation are, however, valuable adjuncts. Pressure upon the upper arm over the area of the radial head produces pain that is very limited, but very acute, and just in the region of the radial head and neck. The pain is increased by attempts at forcible supination. This pain can be easily distinguished from the general pain which any injury produces.

Abnormal mobility and crepitus are signs of little value, as they are frequently wanting. The movements that can be made by the patient are of prime importance. Supination to any extent is always impossible. This is, however, true of other fractures, but is a valuable confirmatory sign. One sign is not enough in any fracture. The radiographic examination will confirm these signs and add a precision and completeness to the diagnosis which are of extreme value. The skiagraphs must, however, be made with proper care and by a person who understands the surgical possibilities, or else they may be of little value. They should be taken in more than one position if a fracture is to be excluded. The diagnosis is not easy without the skiagraph, and errors are liable to occur frequently. The confusion can readily be made with fracture of the external condyle and forward luxation of the forearm or radius. Where the fracture and deformity are of long standing the diagnosis would be impossible except for the aid given by the Röntgen method of diagnosis.

The treatment should be very simple: by massage and passive motion. Reduction is often impossible, and it is always impossible to maintain the bones in position, no matter what bandage or splints are employed; while their use is liable to produce a restriction in motion and to destroy the function of the joint; the latter will be maintained by the treatment without bandages or splints. If, in spite of all treatment and massage, there remains functional loss, or this is the result of an ancient and vicious union, the surgeon should not hesitate to cut down and remove the head of the radius. The results of such intervention are usually very good.

Cancer of the Breast.—BANKS (*Lancet*, April 7, 1900), in speaking of the operations for cancer of the breast, says of the very radical and extensive operations, that he does not believe it necessary to remove the sternal portion of the pectoralis major and to divide the pectoralis minor and reflect that muscle in order to remove all the glands of the axilla. If the arm be properly manipulated and the great pectoral well dragged upward and inward the very topmost point of the axilla can be cleaned out. Moreover, if there is any doubt at all, the great pectoral can be cut across with very little trouble and then stitched together with catgut sutures, as he has frequently done. With an experience of 250 operations he has given up attempting to get any good out of cleaning out the supraclavicular region. When cancer has laid hold of the supraclavicular glands it has got such a grip as to be past extirpating. It is like a burning house—when the fire is beginning even some buckets of water will put it out; when it has got to a certain pitch a whole fire brigade will not quench it. It is clear that in both classes of operation the majority of fatal cases die from internal metastases without external recurrence, and against these no operation can protect. The swing of the pendulum of surgical opinion is generally too extreme, and has now turned from too slight to too extensive operating. The author has never seen a recurrence in the small pectoral muscles; in the great they creep into it from the subcutaneous tissues and pectoral fascia. They do not primarily originate in the muscle. There is, therefore, no need for its removal on that ground.

The operation which he advocates is one in which an elliptical incision

wide of the tumor and of the entire breast is made, with an undercutting, so that the subcutaneous tissues are removed over even a more extensive area than the skin; it reaches even to the sternum. The skin should be particularly freely removed on the side on which the seat of the growth lies. He believes its extension is frequently through the skin, and it is in the skin that most external recurrences are found. The axilla should be opened by an incision just under the edge of the great pectoral, and the whole gland and contents of the cavity, as far as they can be held together, carried away in one mass. The importance of this lies in the fact that recurrence in the axilla takes place for the most part not in the glands that are left behind, but in fragments of lymphatic vessels. A good plan is to get hold of the axillary vein, and then there is nothing else to trouble about. One must take matters very patiently, getting everything out of the cavity that can be got and cleaning up the digitations of the serratus magnus. He uses no knife in the axilla, but two very long, strong pairs of dissecting forceps and curved probe-pointed scissors. He makes a drain-hole just above the edge of the latissimus dorsi in the lower flap, and puts gauze drains through it and also in the upper angle of the axilla. He employs asepsis, pouring hot water over the wound frequently during the operation, and sees that the wound is dry of blood.

Surgery in the Presence of Sugar in the Urine.—Fisk (*Annals of Surgery*, April, 1900) reports a number of cases in which he operated successfully in the presence of sugar in the urine. He reviews the literature of the subject and draws the following conclusions: The presence of glycosuria in those individuals who may have surgical diseases does not in itself constitute an absolute contraindication to any and all surgical relief. Very great judgment must be exercised in the selection of cases, in the determination of the kind and extent of the operation to be performed, and the strictest surgical asepsis must be rigidly enforced throughout. Infection, when it occurs, is from without, and is the result of an error in technique; it thus happens the constitutional symptoms become more serious and out of all proportion to the local, generally ending in death. When infection does not occur the operative wounds heal kindly, but slowly, especially granulating wounds. The vascularity of the tissues must be interfered with as little as possible, so that every operation should be planned with this object in mind. This is particularly so in gangrene of the extremities, in which the statistics of Heidenheim, Kuster, and Smith and Durham show most conclusively the necessity of high amputations in these conditions. He is of the opinion that it is better to cut down upon and ligate the artery in gangrene of the extremities rather than to attempt the bloodless amputation by means of the Esmarch band, because of the possible harm to the tissues, especially the bloodvessels, whose vitality is not the best.

A Method of Early Operation in Perityphlitis.—Kocher (*Correspondenzblatt für schweizer Aertze*, April 15, 1900) describes a new method of operating by which the suppuration present in cases of perityphlitis can be immediately relieved without endangering the peritoneal cavity, while the operation can be made radically complete by the removal of the appendix

at an early date and a secondary operation under absolute asepsis, without any danger of infecting the peritoneal cavity.

The method has been successfuly employed for some time by Prof. Kocher, and is advanced as a solution of the problem which is met with in cases where the inflammation is known to be progressing and possibly to have formed pus, but where there is grave danger in operating immediately by the usual methods, since the adhesions already formed may be too slight to permit the operation without grave danger of infecting the general peritoneal cavity. On the other hand, there is a real danger in inaction, as the adhesions may rupture during the rapid increase of the abscess.

Kocher favors immediate operation as soon as the diagnosis has been established. The majority of cases are seen by the surgeon, however, after a delay has resulted in the hands of the general practitioner. It is in these cases that immediate operation is dangerous, and in which Sonnenburg advises extraperitoneal operation, believing that intraperitoneal operation at this time and diligent search for the appendix are very dangerous. Kocher's method consists, in reality, of two operations—the first is extraperitoneal and antiseptic; the second is transperitoneal or intraperitoneal and aseptic.

The first operation opens the abscess within the area of adhesions by the extraperitoneal route. The pus is evacuated and the abscess tamponed with iodoform gauze, and after draining for two days, until the toxæmia and threatened septic condition of the patient has subsided, when the second or radical portion of the operation is undertaken. The drainage opening is packed with iodoform gauze and closed with sutures. The line of the wound is painted over with iodine and then covered with a sterile dressing. The operation in this part has been antiseptic in character. After careful cleansing of the abdomen, with the regular antiseptic precautions, an incision is made as in appendix operations during the interval, the peritoneum is opened under strict asepsis and the appendix removed. In breaking up the adhesions the abscess cavity is naturally opened, only very little exudate, however, is encountered, and the virulence of the bacteria in this is greatly diminished. In many cases no fluid is found, only fibrous adhesions. After the appendix is removed and the stump covered in and all exudate carefully sponged away the abscess cavity can be closed again, as drainage is afforded by the wound of the first operation. The abdominal wound is closed without necessity of placing gauze drainage in it. The upper layers of the wound are, however, allowed to remain open for two days and then closed by secondary suture. The abscess wound is kept open and allowed to heal by granulation. The two wounds are dressed separately; one aseptically, the other antiseptically, conforming to the methods of operating employed.

A valuable feature of this method of operating is that it permits of bacteriological examination of the pus in the abscess, so that the relative virulence of the infection can be determined before the peritoneum is opened at the second operation. It has been found that there is less danger of further infection in cases where the colon bacillus or the pneumococcus is found than where the staphylococcus, the streptococcus, or a mixed infection is present. The bearing of such information upon the second operation is very

evident. In cases of the milder infection the peritoneum can be depended upon to take care of a slight infection during the second operation. In the graver cases the utmost care has to be exercised.

This operation has the advantage of producing an abdominal wound free from drainage areas, which statistics show lessens very greatly the danger of post-operative hernia.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D.,
OF PHILADELPHIA,

AND

THOMPSON S. WESTCOTT, M.D.,
OF PHILADELPHIA.

Tuberculosis of the Genital Tract in Children.—MARTHA WOLLSTEIN (*Archives of Pediatrics*, May, 1900, p. 347) records a very complete autopsy upon the body of a child, two years old, an inmate of the Infants' Hospital on Randall Island. There was no clinical history other than that the child had had measles, followed by persistent cough and emaciation. A purulent vaginal discharge had been noticed for several weeks before death, but no bacteriological examination had been made. Miliary tubercles were scattered throughout the substance of all the lobes of the lungs; the bronchial lymph-nodes were all enlarged and cheesy, not softened; there were a few small tubercles in the liver, many in the spleen. The peritoneal surface of the rectum was covered with a fibrinopurulent exudate and studded with tubercles, which were scattered all over the pelvic peritoneum. In the right broad ligament was a fluctuating mass, 4 x 6 cm. in diameter, distinctly separated from the uterus, and at first sight obscuring the right tube and ovary. This mass contained green pus, and had bent the right Fallopian tube out of its course. The corresponding ovary contained cheesy material. The left tube was dilated to three times its normal size, but the ovary was normal. Both tubes contained cheesy material. The uterus was normal in size, and its mucous lining healthy in appearance; the serous coat contained many small tubercles. The cul-de-sac of Douglas was occupied by a pocket of pus. Some purulent exudate was found in the vagina, preparations from which contained tubercle bacilli in small numbers and groups of cocci not decolorized by Gram's stain. Tubercle bacilli and staphylococci were also found in the pus of both collections in the pelvis; no gonococci were present.

It was clear, both from the gross appearance and the microscopical study, that the oldest tuberculous lesion was one of the bronchial lymph-nodes, the youngest in the lungs, spleen, and liver; the Fallopian tubes, pelvic peritoneum, and right ovary became involved, in the order named, before the other viscera. Seventeen cases of tuberculosis of the female genital tract

in children, including the author's, have been reported since Talamon's. Only one (Demme's) was less than a year old; five were between one and two years; five between two and three; three between four and five; two were six; one was nine, and one thirteen years old. No pelvic abscess is noted in any of the other cases; the present case is unique in this respect.

The order of frequency of involvement of the different organs differs slightly in childhood from that given by Williams for adult life, and is as follows: tubes, uterus, vagina, vulva, ovary, cervix. The lesion may be primary or secondary, nine of each variety having been reported. Primary infection may occur by direct contact of bacilli with the vulva or vagina; or the organism may be carried primarily to the tubes or uterus by the blood, without the appearance of any lesion at the point of entrance. Secondary infection may occur from a primary lesion in any part of the body, and may affect the genital tract directly, as in the case of a year-old baby reported by Coustensoux, where the tracheobronchial lymph-nodes were the oldest seat of the process, and the Fallopian tubes were involved before the peritoneum.

Differential Diagnosis Between Typhoid Fever and Appendicitis.—S. Weiss, of Vienna, in a communication read at the recent meeting of the American Medical Association (*Medical Record*, June 23, 1900, p. 1100), states that if a drop of blood taken from the lobe of the ear be treated with iodine, the red corpuscle in health becomes yellow and the white cells are unaffected. In disease, however, the multinuclear cells are stained, while the eosinophiles are unaffected. This reaction always shows the presence of pus. Cases of appendicitis and perityphlitis could therefore be differentiated, by means of this reaction, from typhoid fever.

Vaccinal Immunity in the New-born and its Intra-uterine Transmission.—Some interesting facts bearing upon this question are presented in an experimental study by Piéry (*Lyon Médical*, May 13, 1900, p. 37) in which are recorded observations upon fifty-nine cases of vaccination practised concurrently upon the mother and her infant. The mothers were vaccinated once, and upon the infants the operation, if unsuccessful at first, was practised a second time.

Of fifty-nine infants, thirty-six were refractory to vaccination, while twenty-three were vaccinated with success. Of the thirty-six mothers whose infants were thus immune, thirty-two showed themselves refractory to vaccination, and thus proved themelves possessed of the same immunity. Only four infants whose mothers had been vaccinated with success after parturition, and were therefore in a state of receptivity at the time of the infant's birth, manifested immunity when subjected to the test. This conclusion strongly confirms the observation of Béclère, Chambon, Ménard, and Coulon, who found in a series of sixty-five mothers and infants that immunity in the infant was manifested exclusively by those whose mothers showed the same insusceptibility. The author believes that his four exceptions to the rule stated by these observers are scarcely numerous enough to invalidate its truth.

If, however, intra-uterine transmission of immunity in the mother accounts for the immunity of the child, it does not follow that all mothers

thus protected are able to transmit a like immunity to their offspring. In the author's statistics it was noted that while thirty-two mothers transmitted immunity to their offspring, seventeen others bore children who were susceptible to vaccinal inoculation. An interesting observation not included in the series concerned a woman vaccinated without success after a twin birth, who transmitted her immunity to only one of her infants, the other presenting a normal pustule after vaccination.

Combining his results with those of previous observers, Piéry finds that transmission of immunity may be observed in 58 per cent. of the cases.

The reason of the inconstance of transmission of immunity is attributed by Béclère and his associates to the existence in certain subjects, immunized to vaccinia, of a certain anti-virulent property of the blood. When the blood of such persons is intimately mixed with vaccine virus the latter loses its active properties. They, therefore, conclude that the transmission of vaccinal immunity to the infants is observed only among women whose blood possesses this antitoxic property.

The intra-uterine transmission of immunity to vaccinia is observed among mothers who have been vaccinated during their early infancy as well as during their pregnancy. Judging from his own cases, the author is disposed to conclude that transmission is more frequently observed among the former class. Of five women vaccinated successfully during the last fifteen days of their pregnancy, and whose pre-existing immunity was therefore disproved, only one transmitted immunity to her infant, which gives a proportion of 20 per cent. as representing the frequency of transmission of immunity acquired in the last fifteen days of pregnancy. On the other hand, of forty-four women enjoying an immunity dating from vaccination practised in their infancy, thirty-one transmitted it to their infants. This would give a proportion of 70 per cent. among the mothers whose immunity was acquired in their own infancy. This observation also bears out the statement of Béclère and his associates, that the antitoxic quality of the mother's blood-serum is manifested when she has acquired immunity either in early infancy or during pregnancy.

In conclusion, the author states that he does not regard pregnancy as a contraindication to vaccination, since he has observed no appreciable disturbance in any of the pregnant women he has vaccinated.

THERAPEUTICS.

UNDER THE CHARGE OF

REYNOLD W. WILCOX, M.D., LL.D.,

PROFESSOR OF MEDICINE AND THERAPEUTICS AT THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL; VISITING PHYSICIAN TO ST. MARK'S HOSPITAL.

Gout and Rheumatism: Etiology and Dietetic Treatment.—Dr. W. H. Porter believes that defective oxidation is a chief predisposing factor resulting in one instance in rheumatism and in another producing gout.

The suboxidation state is due chiefly to the ingestion of oxidizable food products in larger amounts than there is oxygen absorbed through the lungs completely to reduce the proteid constituents to their end-products. Further, diminished food supply of poor quality may cause suboxidation; in this case progressive malnutrition causes anæmia, and with it insufficient oxygen occurs. A discussion of various theories to account for the formation of uric acid follows, with support of the idea that uric-acid production results from oxidation of proteid substances in the protoplasm of the renal cells. When the amount of oxygen taken up by the lungs is diminished, oxidation of the proteids falls to an abnormally low point, the excretion of urea decreases, and that of uric acid increases. If carbohydrates or fats are taken in large amounts, the oxygenating capacity of the body is exceeded, suboxidation results, and uric acid increases in the urine, while urea diminishes. Thus increase of uric acid in the urine is a symptom indicating an imperfect proteid oxidation and general malnutrition. When the renal cells cease temporarily to act, failing to produce uric acid, a vicarious process occurs in the tissues, oxygenation of proteid substances forms uric acid in the tissue cells, and sodium urate immediately results. This more frequently occurs in the cartilage cells of the first metatarso-phalangeal joint. Urate of sodium acts as an intense chemical irritant, exciting a local inflammatory process; a fibrinous exudate is thrown around the deposit, and the local symptoms subside.

Non-nitrogenous food products are directly oxidized to carbon dioxide and water, yielding no toxic bi-products; therefore the proteids are the source of these toxic and suboxidative conditions of the system. Confirmation of the reported observations on a specific germ for rheumatism is lacking. Probably, however, with overfeeding, bacterial action has much to do with many types of suboxidation, through their disturbing influence on digestion. It may be that they are the factors determining whether the form of suboxidation is to be rheumatism or gout. The bacterial action is on the proteid constituents as they exist in the chyme, for the most part. Animal food is more easily digested than vegetable, but this renders the former more prone to suboxidation, unless the amount taken is restricted so that it is kept well within the oxygenating capacity of the system. Vegetable foodstuffs are more difficult to digest, absorb, and assimilate. There is less danger of exceeding the oxygenating capacity of the system on a vegetable diet, because a large proportion passes through the alimentary canal undigested. Thus good results often follow a strict vegetable diet. The large amount of waste material of a vegetable diet often causes diarrhœa. A catarrhal mucous membrane results and offers a good nidus for bacterial growth, with undue putrefactive fermentation. Cane sugar has to be converted to glucose before absorption occurs—an excessive tax on digestive energy. If these unpleasant results do not ensue much benefit may be derived from a vegetable diet. Fruits, as a class, are to be avoided, being liable to cause fermentation.

Best results follow a mixed diet in which animal food preponderates. The total amount of food ingested must never exceed the oxygenating capacity. Suitable medication to augment glandular action (digestion) should be added. —*New York Medical Journal*, 1900, No. 1112, p. 411.

Observations and Suggestions Concerning Hypodermoclysis —Dr. Robert C. Kemp recommends the space between the highest part of the crest of the ilium and the lower border of the ribs toward the outer margin of the lumbar region as the site of election. Dorsal posture is not interfered with, nor do muscular or respiratory movements cause discomfort. Strength of solution, one drachm of sodium chloride to one pint of water. Everything connected with the operation must be aseptic. The specific effect on renal secretion produced by small quantities of normal saline solution appears rapidly. When irrigations were given and potassium ferrocyanide was added to the solution the urine gave a blue reaction with ferric chloride in less than two minutes, and in addition there was increase of urinary flow. If hypodermoclysis be practised a similar reaction can be obtained in less than four minutes, with an increase in renal secretion. Enormous increase of urine may follow small rectal injections frequently repeated. Small quantities frequently given subcutaneously act as a better diuretic, causing less strain on the kidneys than a pint thrice daily. By thus increasing the urine excretion of injurious substances is provoked, as in acute diseases. A saline enteroclysis (110° F.) gives the following:

1. An immediate increase in arterial tension. If the temperature of the injection is under 110° F. this result does not follow.

2. At the expiration of ten minutes this stimulation seems to reach its maximum point; arterial tension remains then unchanged.

3. Increase of renal secretion begins after ten minutes, coincident with the increased arterial tension.

4. At the same time there is an increase of blood (?) and body temperature.

5. A second marked increase of renal secretion occurs at the end of twenty minutes' enteroclysis, due to absorption from the intestine. In uræmia, shock, etc., lymphatic absorption is nearly at a standstill; even a small hypodermoclysis requires a long time for absorption; in addition there is no stimulating effect from heat.

Saline solution distributed over a large surface, as the peritoneum, either by enteroclysis or enema, has no resistant tension to overcome. There is a wide-spread area for absorption, together with reflex stimulation of the circulation by heat, and, hence, rapid lymphatic absorption. Thus the action of a hypodermoclysis can be much hastened by a simultaneous hot enema or enteroclysis at 110° to 125° F. In hemorrhage from typhoid and gastric ulcers, hypodermoclysis replaces fluid. It enters the body slowly through the lymphatic system, and is less likely than infusion to cause an increase in hemorrhage. Hypodermoclysis is recommended in various forms of poisoning, especially when the drug is eliminated through the kidneys. In pleurisy with effusion, with renal insufficiency, hot enteroclysis may produce sufficient diuresis to absorb the effusion without aspiration. Then follow recommendations for the use of hypodermoclysis in the colitis of infants, in nephritis complicating diphtheria, with oliguria, in uræmia, and in the preparation for operation of a patient who is suffering from shock.—*Medical Record*, 1900, No. 15, p. 623.

Hepatic Engorgements.—Dr. J. Guerder, studying the subject with reference to their occurrence in pulmonary tuberculosis, sums up his treat-

ment as follows: (1) Revulsions comprise mustard poultices, tincture of iodine, dry or wet cupping, and even blisters. (2) Intestinal antisepsis is obtained with salol or naphtol; but if these are badly borne, daily evacuations should be secured by calomel, or, preferably, podophyllin, rhubarb, aloes, or purified ox-gall in three to five-grain doses. (3) Food which contains toxin should be avoided. Tea, coffee, and alcohol should be forbidden. Milk and its preparations are recommended. With the milk Vichy can be taken. (4) Opotherapy has given good results in two recent instances. Enemata of hepatic extract were employed. Inasmuch as the author looks upon hepatic insufficiency as the predisposing cause of pulmonary tuberculosis, this treatment is important.—*Revue de Thérapeutique Médico-Chirurgicale*, 1900, No. 8, p. 253.

Expulsion of Gallstones by the Use of Sulphur Water.—Dr. Axel Winckler reports an instance of the expulsion of fifteen stones after the use of twelve ounces of sulphur water (Bad Neundorf), aided by sulphur baths. The explanation is that sulphur water increases the secretion of the bile. Neundorf water contains 4.5 per cent. of hydrogen sulphide, and in that respect is the strongest sulphur water in Europe. After the expulsion of the stones the use of the water should be persisted in, so that the bile may be kept fluid.

In this connection attention is called to the fact that extensive use of eggs may readily be the cause of the condition, and a cause frequently overlooked. The yolk contains cholesterin in the amount of one-half of 1 per cent., while gallstones are nearly 8 per cent. cholesterin. Bread and the seeds of cereals contain this substance also, and should be limited in quantity.—*Therapeutische Monatshefte*, 1900, Heft 5, S. 246.

Diabetes Mellitus and its Treatment.—Dr. George D. Barny considers the dietetic treatment of great importance, the object being to exclude sugars, starches, and other glucose-forming bodies. Sometimes a certain small amount of sugar is requisite to maintain the best state of health. Gluten bread is permissible. Almond flour is a fair substitute for wheat flour. Soups can be made without starch. Fish, poultry, game, and meats, except liver; a variety of vegetables containing smaller amounts of starch. Tea and coffee are sweetened with saccharine or glycerin. Diabetics are very susceptible to cold and require a warm environment. The disease is evidence of a grave malnutrition, primarily due to perverted nervous function. The liver performs much of its normal work, but it loses the control of the glycogenic centre. Glucose is formed more rapidly, and there is an increase in the destruction of the red blood-corpuscles. Intestinal bacteria and unchanged peptones from the stomach enter the blood. All tissues lack sufficient food. Indications for treatment are to regulate the nervous influence, improve the blood and circulation, prevent the invasion of micro-organisms and inhibitory products. The double bromide of gold and arsenic combines the tonic alterative elements of the two metals and the sedation of bromine. It restores the integrity of the glycogenic centre; it increases the vascular tone and limits the amount of blood supplied to the liver. Digestion is improved, and nutrition likewise. The quantity of sugar is notably diminished;

thirst likewise. Mental depression disappears. It should be given after meals, beginning with five drops, and increasing one drop daily until toleration ceases; toxic symptoms are as for arsenic. Patients vary; some will take only ten drops, and others thirty or forty. Appended are urinalyses from three patients. In all the quantity rapidly diminished to far below normal (twenty-seven ounces). Specific gravity from over 1030 to 1017. There was no albumin present at any time. Sugar, which was present (more than 1.5 per cent. in each), disappeared wholly after ten weeks, with no subsequent recurrence. Weight was not affected. Weekly blood examinations were made in two instances, and showed secondary anæmia, which improved in all respects under the above treatment.—*New York Medical Journal*, 1900, No. 13, p. 463.

Hyoscine.—Dr. Henry S. Noble, after many years of constant use of this drug among the insane, has reached the following conclusions: (1) All patients do not behave the same under its administration. In acute or recurrent excitement two or three of every five will be benefited, and in the recurrent form of insanity attacks of maniacal excitement are frequently averted. (2) Old people, particularly in feeble health, are more profoundly affected by even moderate doses. (3) It is just as efficacious by the mouth as hypodermatically, although its action is not quite so rapid. (4) A tolerance is not established except within limits. If, after increasing the dose to one-sixtieth of a grain, the desired effect is not obtained, it is useless to increase it further. (5) Frequent doses are not required; two in twenty-four hours being usually sufficient. (6) It does not act as a hypnotic except in a peculiar way, wholly different from chloral, opium, cannabis indica, sulphonal, or paraldehyde. It produces sleep normally by allaying cerebral excitement and morbid motor activity. It may be combined with potassium bromide or chloral. (7) Nothing like curative effects have been obtained. Instances of recurrent insanity and *folie circulaire* may be kept in the convalescent wards by its judicious use which otherwise would have spent fully one-third of their time in the excited wards. Its tastelessness permits it being introduced surreptitiously into the patient's coffee should he refuse to take his medicine. For the initial dose one-hundredth of a grain is sufficient.—*Yale Medical Journal*, 1900, No. 8, p. 323.

A Practical Method of Administering Trional to the Insane.—Dott. Corrado Ferrarini employs an effervescent magnesia solution, which not only is a convenient method, but also permits a smaller dose. An effective dose varies from seven to fifteen grains; its more easy absorption in the presence of carbon dioxide is the reason for the smaller dose. Further, when thus administered gastric and intestinal disturbances, so readily excited in the insane, are avoided.—*La Riforma Medica*, 1900, No. 109, p. 402.

The Treatment of Alcoholism.—M. Crivelli has obtained much relief and produced many permanent cures by the following method: At the outset injections of artificial serum, later absolute rest, baths, massage, limited diet, coffee, and finally hypodermatic injections of strychnine in 1 per cent. solution, commencing with three drops twice daily, and increasing two drops

each day until the appearance of the first symptoms of poisoning, which generally occur after twenty to forty drops, representing three to five sixty-fourths of a grain. The dose is then gradually reduced to the initial one.—*La Progrès Médicale*, 1900, No. 19, p. 295.

GYNECOLOGY.

UNDER THE CHARGE OF

HENRY C. COE, M.D.,
OF NEW YORK.

Palliative Operation for Inoperable Carcinoma Uteri.—KÜSTNER (*Centralblatt für Gynäkologie*, 1900, No. 14) recommends the following procedure, which he has adopted successfully in cases of advanced cancer of the cervix. After thorough curettement and cauterization of the diseased area a tampon saturated with alcohol is applied to the raw surface. A large transverse opening in the recto-vaginal septum above the sphincter ani is made, and its edges are sutured with catgut. A long thread attached to the tampon is carried through the fistula and brought out at the anus.

Kolpokleisis is next performed, the vulva being closed with sutures of silkworm-gut, the denuded surfaces being made as broad as possible. The tampon is withdrawn through the anus on the fourth day. As the external wound may not heal perfectly, additional sutures may be required subsequently. It is advisable to irrigate the vagina occasionally through the fistula and to dilate the latter with the finger if it becomes contracted. In case of hemorrhage the vagina can be tamponed (through the fistulous opening?) or irrigated with cold (?) water.

The writer states that patients are considerably benefited by this operation, being relieved of the constant acrid, foul-smelling discharge.

[The writer does not appear to take into consideration certain dangers which may follow closure of the vagina, especially that of septic infection from the retention of discharges and fecal matter in the pocket below the fistula. The difficulty of arresting a profuse hemorrhage from the ulcerated surface after kolpokleisis has been performed may readily be imagined.—H. C. C.]

Specific Micro-organisms in the Female Urethra.—SCHENK and AUSTERLITZ (*Wiener klinische Wochenschrift*, 1900, No. 14), from bacteriological examinations in sixty normal cases, found no germs whatever in one-half and pathogenic germs in only two instances. Savor found in 93 gynecological patients who had not had gonorrhœa that germs were present in the urethra in 59, staphylococcus pyogenes in 22, and colon bacteria in 14. In 120 pregnant women the urethra contained the same varieties of micro-organisms in all but two cases. The writers arrive at a different conclusion, believing that pathogenic germs are rarely found in the normal female urethra, and

that none are present in over 50 per cent. of cases of pregnant and puerperal women. Most of these germs are saprophytes.

Steam in the Treatment of Endometritis.—JOHNSON (*Boston Medical and Surgical Journal*, 1900, No. 11) reports thirty-one cases in which he used this agent successfully. He introduces steam into the uterus from an ordinary throat atomizer (after the cervix has been dilated with thorough aseptic precautions) through a hard-rubber tube, which is removed after thirty seconds, the uterine cavity wiped out with gauze, and the steam again introduced for thirty seconds. The patient is kept in bed from four to six days. The temperature of the steam is never above 212° F.

The advantage of steam over the curette lies in the fact that it acts only on diseased surfaces in puerperal septic cases, while in cases of chronic hyperplastic endometritis the entire endometrium is affected, while even after thorough use of the curette islands of diseased tissue are often left.

Exact clinical evidence was obtained by the author by removing uteri after they had been steamed for different periods and examining them microscopically. It was found that after introducing the steam in the manner above mentioned the endometrium was destroyed, but the muscular tissue was not affected, as was the case when the operation was prolonged beyond a minute. With a longer exposure there is considerable risk of subsequent obliteration of the uterine cavity.

Torsion of the Fallopian Tube.—HARTMANN (*Comptes Rendus de la Soc. d'Obstétrique de Gyn. et de Pædiatrie*, February, 1900) adds five cases to the ten previously reported. In seven of these the tube alone was twisted, while in five the ovary shared in the torsion. In ten the right tube was affected. In some cases the tube was previously diseased, while in others the pathological changes present (especially hemorrhages) were due to the torsion. The vessels in the pedicle were nearly always filled with thrombi.

Clinically the symptoms noted were sudden pain, simulating appendicitis or intestinal obstruction; or successive attacks occurred like renal colic, the latter being in cases of gradual torsion. Localized peritonitis and a rapid increase in size of the affected tube were constant.

Obesity as a Cause of Sterility.—PAOLI (abstract of thesis in *La Gynécologie*, February 15, 1900) calls attention to the fact that obesity is often associated with malformations in the genital tract, nervous and vascular disturbances, and errors in secretion. Hence results painful, scanty, and irregular menstruation, infrequent conception, and if this occurs abortion is common. Sterility in fat women has been variously attributed to pelvic lesions and arthritic troubles. Treatment of sterility in such subjects should be reserved for cases in which disturbances of secretion are marked. If congenital malformations exist the prognosis is hopeless.

[The writer has referred rather vaguely to that interesting class of cases in which a rapid increase of adipose seems to be directly associated with suspension of the ovarian functions. That this condition is not due to true atrophy is proved by the fact that menstruation may return and even conception occur after the excessive weight has been reduced by rigid diet and

exercise. This intimate relation between the ovarian function and metabolism would seem to furnish an argument in favor of the so-called internal secretion of the ovary.—ED.]

Metrorrhagia in Young Girls.—SIREDY (*Revue prat. d'Obstétrique et de Gynécologie*, 1900, No. 3) calls attention to cases of metrorrhagia in young girls in whom no local cause can be discovered to account for the phenomenon. The writer believes that while heredity may play some part, the natural tendency is aided by overexertion, especially by horseback riding, cycling, dancing, etc., which stimulate the pelvic circulation.

As regards treatment, hot vaginal douches and tampons are rarely necessary, and should not be resorted to except in extreme cases. Prolonged hot rectal irrigation with the double-current tube is a useful means of local treatment. Absolute rest in bed throughout the entire period should be maintained. Long walks, dancing, the use of the bicycle and sewing machine, horseback riding, or long standing must be interdicted, and at other times should be permitted only in moderation.

Careful attention to the general health, regulation of diet, and the overcoming of inherited defects are important adjuvants. Hydrotherapy, especially the cold douche, is a valuable means of diminishing pelvic congestion. Life in the country, with the absence of all exciting social elements, is preferable.

Calcareous Deposit in the Tube.—PESTALOZZA (*La Settimana Medica; La Gynécologie*, February 15, 1900) describes a small, pedunculated growth, the size of a hazelnut, which he found attached to the end of a Fallopian tube. It contained a calcified nodule. The wall of the opposite tube was generally calcified. The writer regards this case as quite rare, in fact as equalled only by one described by Potaillon.

Pneumonia after Gynecological Operations.—ANOUFRIER (*Journ. russe d'Accouch. et d'Obstétrique*, 1899, No. 9) calls attention to the occurrence of streptococcic pneumonia after unclean vaginal operations, especially extirpation of the cancerous uterus. Saprophytes are carried through the lymphatics and bloodvessels to the lungs, where they prepare a favorable culture-medium for streptococci. Septic pneumonia may develop from wounds without suppuration being present, as in a case cited by the author. He urges more vigorous attention to vaginal asepsis, by spending a week in thoroughly cleansing the canal before operation.

Hysteropexy in a Child.—VILLEMIN (*La Gynécologie*, February 15, 1899) reports the case of a girl, aged fourteen years, with complete procidentia of two years' standing, which developed suddenly while she was lifting a heavy weight. The little patient experienced a sudden, severe pain in the lower abdomen, and noticed a protrusion from the vulva. Shame led her to conceal her trouble until a few days before entrance to the hospital, when a second attack of pain followed another effort at lifting. The cervix, which was nearly two inches long, was amputated, and hysteropexy was performed successfully.

OBSTETRICS.

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE; PROFESSOR OF OBSTETRICS AND DISEASES OF INFANCY IN THE PHILADELPHIA POLYCLINIC; CLINICAL PROFESSOR OF DISEASES OF CHILDREN IN THE WOMAN'S MEDICAL COLLEGE; VISITING OBSTETRICIAN TO THE PHILADELPHIA HOSPITAL, ETC.

The Treatment of Tumors Complicating Pregnancy.—In the *Medical News*, 1900, p. 1028, WELLS contributes a paper upon this subject. He estimates that two-thirds of the cases of pregnancy complicated by the early stages of cancer go on toward term. In spite of this fact, in view of the feeble vitality of children so born, and of the dangers to the mother which birth brings about, the presence of cancer at any stage of pregnancy demands a radical operation. When the body of the uterus has attained some size, he advocates Werder's method. By this the ovarian and uterine arteries are tied through an abdominal incision, the uterus freed from the bladder and broad ligament, without cutting through the abdominal wall. The vagina is then freed from its adhesions by blunt dissection, and the uterus drawn down and out through the vulva. The peritoneum is then united over the fundus, the abdominal wound is closed, and the operation completed by removing the uterus after dividing the inverted vagina at the point selected. If the uterus is too large, a supravaginal amputation should first be made to lessen its bulk before the removal below of the cervix and vagina.

As regards fibroids complicating pregnancy, the gravest cases are those in which the fibroid occupies the pelvic cavity. Abnormal attachments of the placenta, post-partum and puerperal hemorrhages, and other complications may arise. Myomectomy is justifiable with small subperitoneal tumors of the fundus or any sort of fibroids so situated that they can be removed. With interstitial tumors, supravaginal hysterectomy is the operation of choice. During labor attention should be given to securing contraction of the uterus during the third stage. If post-partum hemorrhage occurs the womb should be explored by the hand, as sometimes fibroids are found that can be easily enucleated, and the womb should then be packed with gauze. Ergot should also be used. Where the tumor is at the fundus, inversion of the uterus may result, which necessitates immediate enucleation and replacement or hysterectomy. When the tumor is in the anterior wall of the womb it may sometimes be pushed out of the way with the patient in the knee-chest position. This cannot happen if the tumor is lateral or posterior. Fibroid polyps should be removed after labor to prevent sloughing.

In ovarian tumors complicating pregnancy the tumor should be removed if detected early in pregnancy. After the fifth month, if the cyst is impacted and not large, the interests of the fœtus may be consulted, and operation may be postponed until it is viable. Should, however, impaction or obstruction to labor occur by an ovarian cyst, abdominal section is the better procedure.

The Indications for Premature Delivery with Reference to Eclampsia.—MARX (*Medical News*, 1900, p. 1030) contributes a paper in which he lays special stress upon the toxaemia of pregnancy. He believes that urea is always found greatly diminished in this condition. He makes a strong plea for a methodical course of urea estimation in all cases. He believes that progressive diminution of urea excretion, with or without albuminuria, is the sole indication for the induction of premature labor, which is especially indicated when conscientious medical treatment fails.

As regards the induction of labor for pelvic contraction he believes that the bony pelvis affords an absolute indication in very few cases. He believes that the comparative size of the pelvis with reference to the head is the important matter. If the head fails to engage, it is proof positive that the pelvis is too small for the head or the head too large, and interference must be practised. The pelvimeter is an instrument for comparison and not one of precision.

Gangrene of the Puerperal Uterus.—BECKMANN contributes an interesting and extensive paper upon this subject, with illustrations, in the *Zeitschrift für Geburtshülfe und Gynäkologie*, 1900, Band xlii., Heft 3. In his observation, gangrene of the puerperal uterus is not infrequent. He has found it present in a considerable number of affections of the puerperal uterus, more frequently in private practice than in maternity hospitals, because in the former the frequency of puerperal septic infection is greater.

The diagnosis of this condition is not readily made. There are no definite symptoms which point to this complication. When, however, cases of septic infection are differentiated the diagnosis is not so difficult. The enlargement of the uterus during the first few days of the disease, delayed involution, swelling of the inner surface of the uterine wall, and the expulsion of this tissue are the characteristic phenomena. The temperature curve is that commonly seen in gangrene or pyæmia. The prognosis depends upon the severity of the infection, upon the complications present, and especially upon the presence or absence of perforation of the uterus. The death-rate is stated as 27.5 per cent. Uncomplicated cases usually end in recovery. Whether the uterus resumes its function after the patient's illness depends upon the amount of necrotic tissue and the proportion of uterine surface which is destroyed. The mucous membrane of the womb does not, as a rule, re-form.

Streptococcus infection is the sort usually present in these cases. This spreads through the deeper bloodvessels of the uterus and also through the lymph-vessels, with the formation of thrombi. Necrosis of the connective tissue results, and in severe cases the patient dies before the necrotic tissue can separate. Usually, however, dead tissue comes away with the free formation of pus. In three cases saprophytes were found, although it cannot be definitely known whether the ordinary bacteria of putrefaction are instrumental in producing this condition.

In the treatment 52 per cent. of the cases were subjected to some sort of operative treatment. In some this was instrumental delivery after a prolonged labor. In other cases typhoid infection had preceded the patient's labor. In portions of the uterine tissue which did not become gangrenous degeneration of the muscle-fibre was observed.

In selecting treatment for these cases attention is called to the danger of douching or curetting the uterus.

[In treating such cases the drainage of the womb must be properly secured. If the uterus is retroverted it should be replaced and held in position by a packing of antiseptic gauze. This must be frequently changed and the vagina gently but thoroughly irrigated. The patient's general condition requires very careful and persistent stimulation.—ED.]

Puerperal Eclampsia Treated by Hypodermoclysis, with Diuretic Infusions.—Appreciating the difficulty of securing prompt secretion of urine in eclamptic cases, JARDINE has for three years employed saline infusions consisting of sodium chloride and potassium bicarborate cr sodium acetate (*British Medical Journal*, 1900, p. 1279). The writer controls the fits by using chloroform, veratrum viride, or chloral and bromide; he usually prefers veratrum viride. Whenever possible he administers magnesium sulphate by the mouth, using four to six tablespoonfuls in warm water. If the patient cannot swallow it is given through a tube. Chloral and bromide are given with it in many cases. A hot pack is also employed.

He places, however, the greatest reliance upon introducing a solution of one drachm each of potassium bicarbonate and sodium chloride to two pints of boiled water. From one to three pints will be absorbed at a time. The temperature of the water should be 104° F., and antiseptic precautions should be employed with the needle and in cleansing the skin. The needle is usually introduced beneath the breast. No special apparatus is required, a sterile trocar and canula, tubing, and a funnel being all that is needed. In 200 infusions no case of abscess has occurred.

[We have also employed this method for some time, with the best results. A more prompt and efficient method of using the same agents is found in intravenous transfusion. Added to this we may often practice to advantage copious injections of magnesium sulphate into the intestines, passing a rectal tube as high as possible and using as much fluid as can possibly be retained. We have seen very prompt diuresis follow this method of treatment.]

The Frequency and Significance of Infarcts of the Placenta.—In the recent anniversary publication dedicated by his pupils to Professor Welch, WILLIAMS contributes a paper under the above title.

He has examined 500 placentæ to determine the significance of infarcts, and illustrates his paper by a reproduction of microscopical sections. He concludes that infarcts measuring at least 1 cm. in diameter were observed in 63 per cent. of his cases. The great majority of placentæ have small infarcts just visible to the naked eye, while microscopical examination shows early stages of infarct formation in all full-term placentæ. Endarteritis of the vessels of the chorionic villi is the cause of these infarcts. This results in coagulation necrosis in the villi just beneath the syncytium, with subsequent formation of canalized fibrin. The syncytium also degenerates, and is similarly converted, and blood coagulates beneath the villi, matting together groups of villi by masses of fibrin. Later the stroma of the villi degenerates and the infarct becomes a network of fibrin. In many cases this arises from the fœtal ectoderm rather than from the decidua. Moderate

degrees of this process are not pathological, and have no influence upon the mother or child, but denote senility of the placenta. Marked formation of infarcts often results in death or imperfect development of the fœtus. It is usually associated with albuminuria on the part of the mother. Why, we cannot say. In cases of eclampsia infarct formation is not especially noticeable, but is usually observed only in those cases which were preceded by albuminuric symptoms. There is no evidence that bacteria had anything to do with this process.

DERMATOLOGY.

UNDER THE CHARGE OF

LOUIS A. DUHRING, M.D.,

PROFESSOR OF DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA,

AND

MILTON B. HARTZELL, M.D.,

INSTRUCTOR IN DERMATOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

Urticaria.—PHILIPPSON (*Giorn. Ital. delle Mal Ven. e delle Pelle*, 1899, Fasc. v.; *British Journal of Dermatology*, June, 1900) opposes the current opinion held by most dermatologists, that urticaria is due to reflex nervous action exerted on the bloodvessels; he believes with Haidenhain that a secretory action of the vascular endothelium is involved, and that the œdema which occurs in this disease is similarly produced by direct action of poisonous substances upon the vessels in the neighborhood. He concludes that, just as in the case of erythema, urticaria is a mild inflammation in which the irritant is of low intensity and exerts a more purely local action.

Dermatitis Venenata due to the Common Ivy.—W. J. MUNRO (*Australasian Medical Gazette*, January 20, 1900) reports a case in which an inflammation of the erythemato-vesicular type was set up in a woman, aged forty years, by contact with the wet leaves of the common ivy (*Hedera helix*). In all four distinct attacks were observed, each attack indicating an increased susceptibility to the poison. The lesions were grouped here and there, and bore a resemblance to herpes zoster.

Mycosis Fungoides.—GALLOWAY and MACLEOD (*British Journal of Dermatology*, May and June, 1900) describe three cases and give the following summary of the histological description: In the lesions of the prefungoid stage there exists a connective tissue cell proliferation around the bloodvessels of the subpapillary and papillary layers, the hair follicles, sebaceous glands, coil ducts and occasionally the coil glands, and forming foci independent of these structures situated among the connective tissue bundles. In the epidermis active mitosis of the prickle cells and downgrowth of the interepithelial processes is noted; also nests of corium tissue in the mucous layer,

and interepithelial œdema going on to the formation of reticular spaces. In the tumor stage the cell proliferation increases, and the cells show a marked tendency to break down, as evidenced by the crenation, irregularity, and fragmentation of the cells. The granuloma encroaches on the downgrowing epithelium, flattens it out, spreads up to the surface, and is covered only by a layer of the stratum corneum.

The diseases with which mycosis fungoides has been confused histologically are the granulomata of tubercle and syphilis, the sarcomata, and the leukæmic and pseudoleukæmic growths of the skin. It resembles syphilis in many particulars. In contradistinction to tuberculosis of the skin, giant cells with central caseous degeneration do not occur in mycosis fungoides. In the skin lesions of leukæmia there is great œdema affecting the vessels of the cutis, and active diapedesis from them, and an infiltration of the neighboring cutis with leucocytes; it is purely a leucocytic infiltration without marked fixed-cell proliferation, mitosis, or imperfect giant-cell formation, such as occurs in mycosis fungoides. Pseudoleukæmia cutis is more like a syphilitic granuloma than mycosis fungoides. The bacteriological examination was negative.

An Epidemic of Impetigo Contagiosa.—Ohmann-Dumesnil (*St. Louis Medical and Surgical Journal*, June, 1900) reports thirteen cases that were traced from one individual to another, thus showing contagion. With one exception all occurred in infants or children. The cases were observed for the most part in several families. Antiseptic ointments of camphor and carbolic acid were successfully employed. The author calls attention to the point that the diagnosis should be made early, and treatment instituted before opportunity for spreading has been given.

Pemphigus of the New-born.—Bloch (*Archiv. für Kinderheilk.*, vol. xxviii., 1900) distinguishes a benign and a malignant form, the latter resembling foliaceous pemphigus and also Ritter's exfoliative dermatitis, and are nearly always fatal. It represents a generalized infection, due in most cases to streptococci, the mode of infection being not always clear. It is contagious, and is often spread by the midwife. The article is based on twenty cases, studied clinically, anatomically, and bacteriologically.

Gelatin Applications.—Herbert Skinner (*Brit. Journ. of Derm.*, May, 1900) experimented with gelatin and agar-agar, but the latter was not found useful for the purpose. Gelatin is more tractable, but any formula given will depend on the gelatin used, no two samples seeming alike, while half an hour's treating will make two samples from the same lot behave differently.

A Cream of Carbonate of Zinc.—Herbert Skinner (*Brit. Journ. of Derm.*, May, 1900) prefers carbonate of zinc to commercial calamine, which is often impure and far from desirable as an application to an inflamed surface. The carbonate of zinc compounded as follows makes a useful so-called cream. ℞. Glycerin amyli, adeps. lanæ. hydros., zinci carb, glycerin, of each ℥ss. The first two are mixed together, then the second two, finally both together. The zinc may be increased or decreased.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

W. T. COUNCILMAN, M.D.,

SHATTUCK PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY,

AND

F. B. MALLORY, M.D.,

ASSISTANT PROFESSOR OF PATHOLOGICAL ANATOMY, HARVARD UNIVERSITY.

Elastic Tissue in the Uterus and Ovary.—WOLTKE (*Ziegler's Beiträge*, 1900, vol. xxvii., p. 575), in the investigation of the elastic tissue of the uterus, examined twenty cases ranging from four months to eighty-six years in age. Sections were cut in such a way as to include both serosa and mucosa, and were stained first by Weigert's method, then washed in 96 per cent. alcohol, and placed for a few seconds in a mixture of 1 to 2 per cent. fuchsin solution and a 10 per cent. aqueous solution of picric acid.

There are three muscle layers in the uterus. The outer and inner consist of muscle bundles running longitudinally; the middle layer shows a circular arrangement, and is the thickest. The outer and inner layers are known as the stratum subserosum and stratum submucosum, the middle as the stratum vasculosum. Only in the interstitial tissue of the outer and middle layers of the corpus and fundus uteri are elastic fibres invariably found.

In the middle layer the fibres fail gradually as they run inward, so that they are lacking in the inner layer. They are most numerous in the outer layer, where they surround the muscle bundles and send fine processes in among the muscle fibres. Elastic tissue separates the muscle bundles of the outer layer from the connective tissue serosa, also the subserosa from the serosa of the peritoneum. This condition holds from the age of four months to the climacteric.

After the age of seventy years two changes appear. The elastic fibres in the middle layer form bunches around the bloodvessels, which group themselves together and show signs of sclerosis, and the number of elastic fibres in this layer decreases. These changes are especially marked in senile atrophy at from eighty-three to eighty-six years.

In youth the number of elastic fibres depends on the sexual life. If conception has taken place the number is much increased; in a virgin uterus the number is small. In young people the elastic fibres in the muscularis are thin and delicate; with increasing age they become thicker, and after fifty or sixty years they present a bunched and crumpled appearance. At a still greater age the fibres run together in clumps. The arrangement of fibres about the openings of the tubes is the same as in the body and fundus.

In the portio vaginalis the elastic fibres form a network just under the stratified epithelium, reaching to the mucosa of the cervix. A more deeply lying network surrounds the vessels, and is connected with the superficial layer by slender processes which fail toward the mucosa of the cervix. After seventy years the deeper network disappears.

In inflammation, with hyperplasia of the connective tissue, there is an increase in the elastic fibres. In fresh catarrhal inflammation of the vagina the round cells force themselves between the stratified epithelium and the superficial elastic network, pushing the latter away, and they may even break through the network. If the inflammation becomes chronic, elastic fibres appear in the newly forming fibrous tissue.

In a three-months' pregnant uterus, with the ordinary hyperplasia of the uterine tissue, the elastic fibres increase only in the layers in which they are normally found; they preserve their physiological limits.

In proliferation of the intima of the vessels of the stratum vasculosum large numbers of delicate elastic fibres appear, and the elastica is somewhat thickened. Elastic tissue disappears toward the decidua, and in the spongy layer the bloodvessels have no elastica. In the vaginal portion the elastic tissue is much more abundant in the pregnant than in the virgin uterus. In a word, in the first half of pregnancy the increase in elastic fibres follows the increase in bloodvessels.

In a seven-months' pregnant uterus the elastic fibres are few in number, having rapidly diminished since the third month. The elastic tissue increases with the hyperplasia of the uterus during the first half of pregnancy, and during the last half, as the uterine walls become thinner, the elastic tissue disappears. The vagina shows the same phenomenon, but in the paracervical tissue the elastic fibres are extremely numerous.

In the chorionic villi and septa of the placenta no elastic fibres have been seen.

Examination of the puerperal uterus showed that the elastic fibres regenerate rapidly. As new muscle fibres appear the elastic tissue also regenerates, and delicate fibres can be seen surrounding the muscle bundles and sending processes between the muscle fibres.

The cortical substance of the ovary contains no elastic tissue, but among the vessels and nerves of the hilus elastic tissue is richly developed. In the medullary substance the amount depends on age and on physiological function. In a nineteen-year-old virgin elastin appears only in the vessel walls, but in a primipara of twenty-three years elastic tissue appeared in the medullary substance near the follicles. In youth the fibres surround part of the obliterated follicle, in middle age the whole, and in old age the fibres are thickened and bunched.

In a primipara of twenty-five years it was observed that in the connective tissue which replaced the Graafian follicles the ground substance consisted of innumerable elastic fibres, which formed a delicate network. On the other hand, in another similar ovary no elastic fibres could be demonstrated. However, in most cases elastic tissue can be made out, and it seems to arise from the proliferating ovarian stroma. Up to a certain age the corpora fibrosa increase in number, and about them there is an increase in elastic tissue.

The elastic tissue of the ovarian bloodvessels undergoes changes chiefly in arterio-sclerosis. The elastic fibres in the intima increase in number, and the elastica interna often divides into two layers. If calcification also occurs the media contains little or no elastic tissue. In this case the increase of the fibres in the intima may be considered as a compensatory change to maintain the elasticity of the vessels.

The lumina of the vessels in the medullary substance often become obliterated by the proliferation of cells which adhere to the degenerated walls. The material of the degenerated walls can be seen to consist of thick elastic fibres which have become somewhat hyaline.

In the neighborhood of the corpora fibrosa are bloodvessels which have evidently been obliterated for some time. This is due to newly formed connective tissue which contains much elastic tissue.

Squamous-celled Carcinomatous Degeneration of an Ovarian Dermoid Cyst.—PETERS (*Bulletin of the Johns Hopkins Hospital*, 1900, vol. xi., p 78) gives a detailed description of a specimen in which an epidermoid carcinoma originated in a dermoid cyst of the ovary. Physical examination of the patient did not reveal carcinoma in any other part of the body. The tumor invaded the wall of the cyst and the surrounding tissues. A transition from the lining epithelium of the cyst into the epithelioid cells of the carcinoma was demonstrated. Some of the tumor cells were multinuclear, and their appearance suggested direct division. Giant cells similar to those often met with on the inner surface of dermoid cysts, free from any malignant new-growth, were present. These have been described by Hildebrandt as foreign-body giant cells due to the presence of hairs in the walls of the cysts. Peters, however, has often observed them in parts of the walls of dermoid cysts in which there were no hairs to be seen, and also in simple dermoids in which no hairs were found in any part of the tumor. He believes, with Cullen, that they are more probably a form of embryonic epithelium from which the lining epithelium of the cysts is developed.

A detailed description of the specimen and the clinical and post-operative history of the patient are given.

In reviewing the literature Peters finds that as yet no case of carcinoma thought to be derived from the coil or sebaceous glands of a dermoid cyst has been reported.

Among secondary changes, other than carcinomatous, which have been observed in dermoid cysts, he notes degeneration of glands in the wall of a dermoid and the development of sarcoma and of endothelioma. He reports also a case of carcinomatous degeneration of a part of an ovary in another part of which was a dermoid cyst.

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All communications should be addressed to

DR. ALFRED STENGEL, 1811 Spruce Street, Philadelphia, U. S. A.

Or

DR. HECTOR MACKENZIE, 59 Welbeck St., Cavendish Sq., London, W., Eng.

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